VOLUME 7 NUMBER 3

AMERICAN JOURNAL OF DISEASES OF CHILDREN

EDITORIAL BOARD

[illegible]

MARCH, 1914

PUBLISHED BY
AMERICAN MEDICAL ASSOCIATION
CHICAGO, ILL.

Entered as Second-Class Matter, January 4, 1911, at the Post-Office at Chicago, Illinois, Under Act of Congress of March 3, 1879.

CONTENTS OF VOLUME VII

JANUARY, 1914. NUMBER 1

PAGE

LEUKOCYTE COUNTS IN PNEUMONIA AND CEREBROSPINAL MENINGITIS. JULIUS H. HESS, M.D., CHICAGO 1

DISSEMINATED MILIARY TUBERCULOSIS OF LUNGS AND SKIN. W. P. NORTHRUP, M.D., NEW YORK ..... [illegible]

A CONSIDERATION OF TARDY SYPHILIS. H. H. YERRINGTON, M.D., AND FLORENCE M. HOLSCLAW, M.D., SAN FRANCISCO 22

TREATMENT OF RHEUMATIC ENDOCARDITIS FROM AN OUTPATIENT DEPARTMENT STANDPOINT. WILLIAM P. LUCAS, M.D., SAN FRANCISCO, AND MARK H. WENTWORTH, M.D., BOSTON 41

NEUROTIC VOMITING IN AN INFANT. WM. B. MCCLURE, M.D., BALTIMORE 48

RÉSUMÉ OF WORK ON TUBERCULOSIS IN CHILDREN FOR 1913. MAY MICHAEL, M.D., CHICAGO 54

INDEX TO CURRENT PEDIATRIC LITERATURE 71

American Journal of Diseases of Children

Vol. 7 MARCH, 1914 No. 3

A STUDY OF THE NUTRITIVE VALUE OF SOME PROPRIETARY INFANT FOODS *

RUTH WHEELER AND ALICE BIESTER

URBANA, ILL.

This investigation was suggested by the accumulating evidence that there may be danger in a restricted dietary even when the latter is entirely satisfactory in total energy value and in the proportion of protein, fat, carbohydrates and essential salts present in it.[1] Many of the proprietary infant foods now frankly advertise that they are only partial foods and as such they often prove of value; but not infrequently they form the chief source of nourishment of infants, and medical literature contains numerous records of malnutrition attributed to them. It is of interest to know whether the failure of such a ration to maintain health or to sustain proper growth is due (*a*) to the character of some of the nutrients, for example, because its proteins can not supply all of the nitrogen requirements of the organism; or (*b*) to the absence from it of some essential "vitamine" or "growth hormone."

If the difficulty is due to the lack of a "vitamine" the addition of a small amount of fresh milk to the ration might correct the defect; if it lies in the character of the nutrients of the food, it might be necessary to make milk the chief part of the diet and to use the food powder merely as a diluent, as is often advised. In either case the question remains open whether milk alone properly modified would not be quite as satisfactory as well as much more economical. Food powders even when made wholly or partly from milk are not necessarily safe as an exclusive diet, for Holst and his coworkers[1] found that drying (in grains) destroys some substance whose absence causes in guinea-pigs a condition resembling scurvy.

---

* From the Research Laboratory of the Department of Household Science of the University of Illinois. Most of the data in the paper are taken from the thesis submitted by Alice Biester for the Master's Degree.

* Submitted for publication Jan. 15, 1914.

1. Neumann: Deutsch. med. Wchnschr., 1902, p. 628; Stepp: Ztschr. f. Biol., 1911, lvii, 156; Suzuki, et al.; Biochem. Ztschr., 1912, xliii, 89; Funk: Jour. Physiol., 1912, xliv, 75; Hopkins: Ibid., p. 425; Holst and Frölich: Ztschr. f. Hyg., 1912, lxxii, 1; Fürst: Ibid., p. 121; Frölich: Ibid., p. 155; McCollum and Davis: Jour. Biol. Chem., 1913, xv, 167; Osborne and Mendel: Ibid., p. 311.

PLAN OF INVESTIGATION

The plan of the present investigation was to feed typical proprietary infant foods to white mice, not cooked or mixed with milk, as such foods are commonly given to infants, but as nearly as possible as they come from the manufacturers, with only such modifications as are necessary to adapt the food to the species of experimental animal chosen: in other words, to study the *nutritive value of the foods in themselves.* With the results of these experiments as a basis, we are hoping to observe the effect of other modifications. The work is only fairly started but some of the facts already apparent seem worth reporting.

Four typical foods have been studied; two, Horlick's malted milk and Nestlè's food, made from milk and malted grain; and two, Mellin's food and Eskay's albuminized food, from grains without the addition of milk. As no recent analyses could be discovered, protein (N x 6.25) and ash determinations were made in each of the four, but the values found

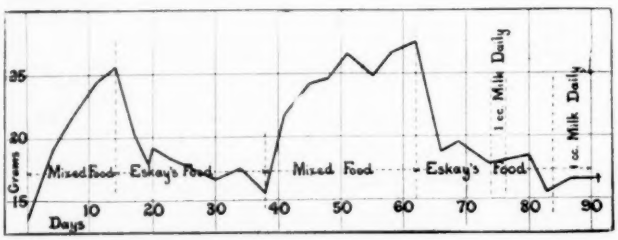

Chart 1.—Mouse 64 ♂, showing loss of body weight in an adult mouse during two periods in which unmodified Eskay's Albuminized Food and water was the only nourishment given. The addition of 1 c.c. of fresh milk daily to this ration at one time for two days and at another time for six days arrested the decline.

varied so little from the figures previously published[2] that we concluded that the process of manufacture had not been materially altered since the analyses cited were made, and availed ourselves of averages for the percentage of the various nutrients present in the foods. Necessary modifications were secured by the addition of purified casein, and, where it was necessary, of milk salts in the form of Osborne and Mendel's[3] protein-free milk (this involved the addition of large quantities of lactose as well). The few data available suggested two formulae[4] for the composition of food for mice: one containing 18 per cent. protein and 4 per cent. ash on which these animals had maintained constant body weight for six months, and one containing 30 per cent.

2. Boyden: Bull. 59, Lab. Int. Rev. Can.; Conn. Ag. Sta. Rept., 1908.

3. Osborne and Mendel: Pub. 156, part II, p. 80, Carnegie Institute of Washington.

4. Wheeler: Jour. Exp. Zool., 1913, xv, 218.

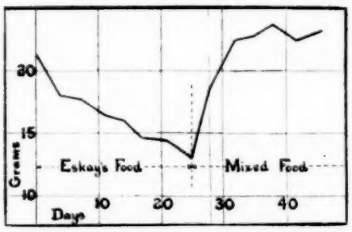

Chart 2.—Mouse 65 ♂, showing loss of weight in an adult mouse on unmodified Eskay's Albuminized Food and recovery on a mixed diet.

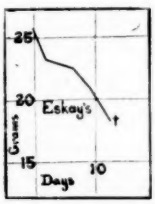

Chart 3.—Mouse 7 ♂, showing loss of weight and death on unmodified Eskay's Albuminized Food.

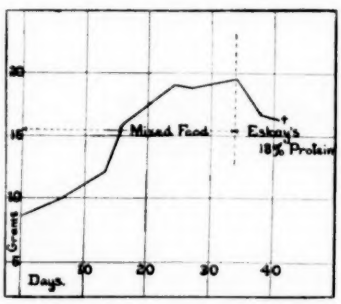

Chart 4.—Mouse 112 ♀, showing loss of weight and death on Eskay's 18 per cent. protein food.

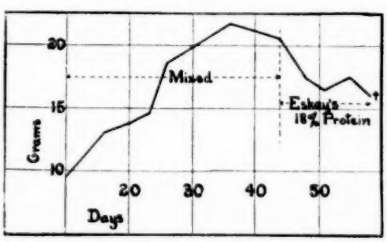

Chart 5.—Mouse 113 ♂, showing loss of weight and death on Eskay's Food modified to contain 18 per cent. protein. (For composition see table.)

protein and 6.6 per cent. ash on which young mice had shown normal growth.

Accordingly, three series of feeding trials were undertaken, the unmodified foods being fed as well as the two modifications mentioned above. All food mixtures were made into a thick cream with water. Control animals were given a general mixed diet. As far as the stock on hand permitted, each experimental group consisted of one male and two females in order that the effect of the diets on reproduction as well as on maintenance and on growth might be observed.

The composition of the food mixtures used is shown in the accompanying table:

Formula	Food A*			Food B			Food C				Food D		
	I	II	III	I	II	III	I	II	III	IV	I	II	III
Food	100.0	67.0	20.0	100.0	97.0	25.0	100.0	90.0	20.0	95.0	100.0	71.0	17.5
Casein	.....	13.0	28.3	.....	3.0	25.5	.....	6.5	27.2	5.0	.....	9.4	27.7
Protein-free milk	.....	20.0	42.5	.....	....	37.0	.....	3.5	39.5	.....	.....	19.6	43.0
Lard	.....	. ..	9.2	.....	....	12.5	.....	.....	13.3	.....	.....	....	11.8
Composition													
Protein	6.7	17.9	30.6	15.3	17.8	30.1	11.9	17.0	30.4	16.0	11.2	17.8	30.6
Salts	1.4	4.0	6.8	3.9	3.7	6.6	3.9	4.0	6.8	3.7	1.6	4.1	6.9
Carbohydrate	87.8	74.8	51.5	75.6	73.4	48.5	81.3	76.0	47.8	77.0	81.1	73.2	48.5
Fat	1.2	.8	9.5	3.5	3.4	13.4	.7	.65	6.89	.69	4.5	3.2	12.6
Calories in 100 gm. (computed)	398	387	424	404	404	447	384	397	387	362	420	403	441

* Food A: Eskay's Albuminized Food; B: Horlick's Malted Milk; C: Mellin's Food; D: Nestlè's Food.

Column 1 under each food = unmodified foods; Column 2 under each food = "18 per cent. protein" modification; Column 3 under each food = "30 per cent. protein" modification.

RESULTS

Each series of feeding trials is discussed by itself because the modification of the foods complicates the problem somewhat by introducing other factors than the intended change in gross composition (if we may use this term to signify percentage of protein, fat, carbohydrate, and inorganic salts, disregarding the presence of traces of other possibly essential substances). Funk[5] suggests that Osborne and Mendel's protein-free milk may contain some of the "vitamine" of the milk; and Hopkins[6] reports that extraction with alcohol and ether removed some

5. Funk: Biochem. Jour., 1913, vii, 211.
6. Hopkins and Neville: Biochem. Jour., 1913, vii, 97.

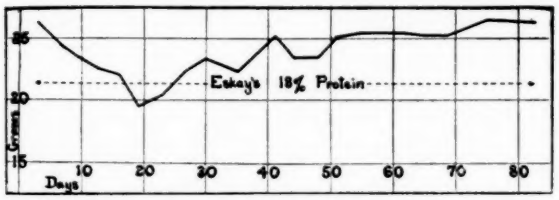

Chart 6.—Mouse ♀, showing initial decline and later repair and maintenance on Eskay's Food modified to contain 18 per cent. protein. (For composition see table.)

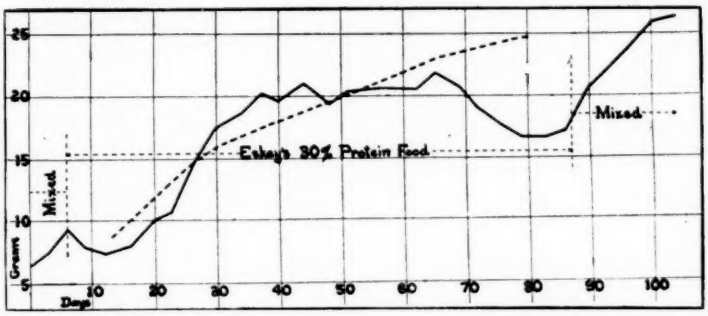

Chart 7.—Mouse 89 ♂, showing normal growth for sixty-one days on Eskay's Food modified to contain 30 per cent. protein. (For composition see p. 172.) After sixty-one days there was a rapid loss of one-fourth the body weight.

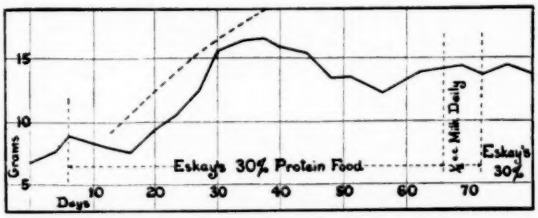

Chart 8.—Mouse 92 ♀, showing nearly normal growth for eighteen days, followed by a slow decline in weight.

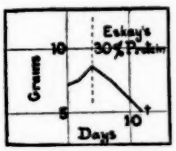

Chart 9.—Mouse 80 ♂, showing decline and death on Eskay's Food modified to contain 30 per cent. protein.

essential substance (vitamine?) from casein as usually purified. Because of these considerations, the extent to which the results of the first series, in which the unchanged foods were studied, parallels those of the other two series, is of especial importance.

Series I—Unmodified Foods

Both of the foods which contained milk incorporated in them in the process of manufacture proved sufficient during relatively long periods to maintain adult mice in health without significant loss of body weight. (Charts 10, 11, 25 and 26). The feeding of Mellin's Food and of Eskay's Albuminized Food containing no milk resulted in a steady loss of weight (Charts 1, 3 18 and 19). These results in themselves do not of course justify the conclusion that milk or some of its constituents is the significant factor, for the foods differ rather widely in composition. A comparison of the results of analyses of Horlick's, Mellin's and Nestlè's Food as shown in the columns marked 1 in the table show, however, that gross composition is not likely to afford the sole explanation, for in every respect except total energy value and fat (which will be considered later) the inadequate Mellin's Food stands between the other two, both of which proved satisfactory. Little inference can be drawn from this series by itself, but

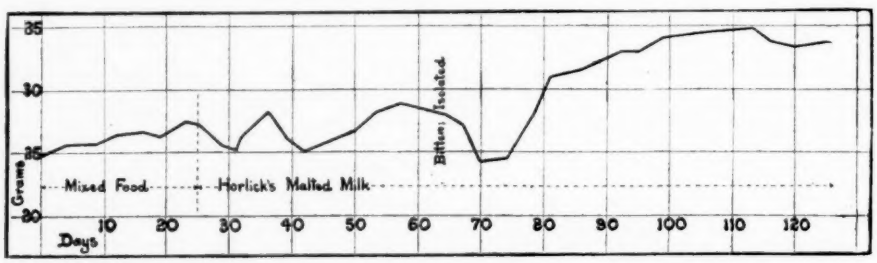

Chart 10.—Mouse 54 ♂, showing maintenance for 100 days and growth to maximum weight in a mature mouse on an exclusive diet of unmodified Horlick's Malted Milk.

Series II and III afford considerable confirmation of the differences in the foods seen here. A more detailed discussion follows the statement of the results of feeding the two modifications (Series II and III) in which it was possible to secure in the four foods more nearly equal gross composition.

Series II—18 Per Cent. Protein Foods

The formulae and the composition of these mixtures are given in the columns marked II in the table. The results of the feeding trials were surprising. As has been said, all we had reason to expect of this modification was maintenance, for on mixtures of purified food substances of similar composition mice had never shown more than minimal growth, seldom even that. (Wheeler.[4] This was confirmed in this laboratory in 1912-13). In no case had there been normal reproduction; in few cases sexual activity.

Horlick's Malted Milk; 18 Per Cent. Protein Modification.—This was most satisfactory. Two mature mice (Charts 12 and 15) lived on it exclusively for more than four months, increased their weight somewhat, and generated two litters each of six young, apparently normal in every respect. The first litter was divided after the suckling period, half being given a general mixed diet and the rest the 18 per cent. protein modification of Horlick's Malted Milk.

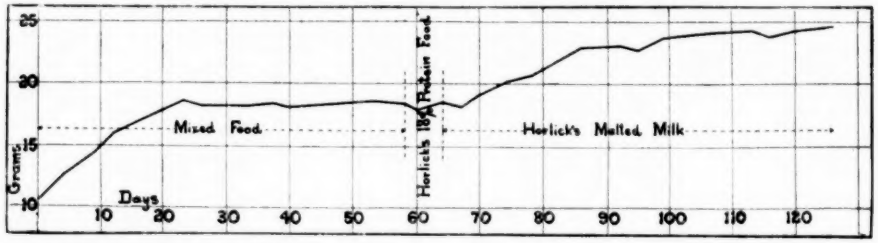

Chart 11.—Mouse 34 ♂, showing growth in a mature mouse on unmodified Horlick's Malted Milk.

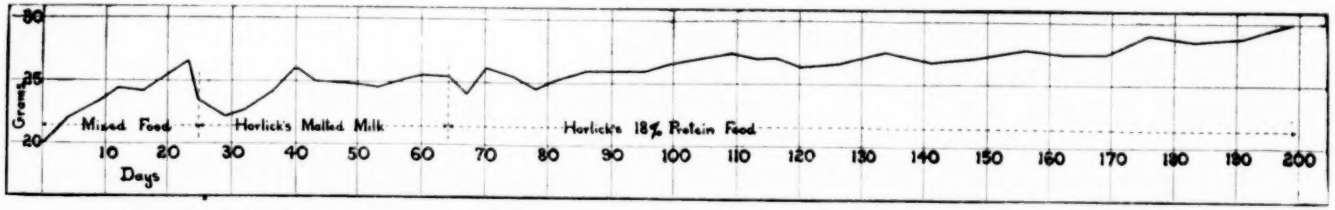

Chart 12.—Mouse 53 ♂, showing maintenance and growth to average maximum weight for 174 days in a mature mouse on Horlick's Malted Milk. For thirty-nine days this animal had the unmodified food; for the subsequent 135 days the 18 per cent. protein modification.

The former grew normally, the latter even more rapidly. A female in each group of this second generation brought forth young which grew normally during the suckling period, at the end of which the experiment had to be terminated. This third generation (of eight mice) were descended from parents which had had no food during their entire life time, except the 18 per cent. protein modification of Horlick's Malted Milk and from grandparents which had only this food for twenty-eight days before the birth of the young. As far as a four months' experiment can prove it, this food mixture seems to be a perfect food (for mice at least) whether considered from the standpoint of maintenance in adults, of growth in the young, or of reproduction. The radical difference between the results here recorded and those previously obtained with mixtures of purified food substances of closely similar composition suggests the presence in Horlick's Malted Milk of small quantities of some "accessory" substance important in growth and in reproduction, although there is still the possibility that maltose is an important factor.

Nestlè's Food; 18 Per Cent. Protein Modification.—We were unfortunate in our choice of animals for the study of this mixture. Both mice seemed entirely normal at the time the experiment was begun. Possibly they were normal at

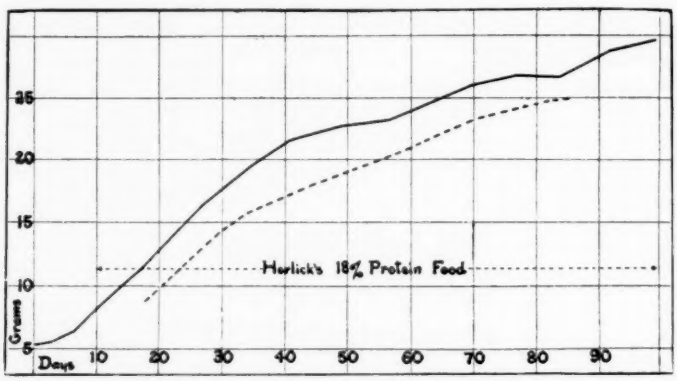

Chart 13.—Mouse 143 ♂, showing growth more rapid than the "normal" on the 18 per cent. protein modification of Horlick's Malted Milk.

that time, but on autopsy two months later, the male proved to have a large parasite encysted in the liver and the female to have diseased lungs. Even with these handicaps the two maintained their weight for more than two months and the female bore a litter of six, four of which lived to the end of the experimental period, gaining weight steadily though at less than the normal rate. (The other two died soon after birth). At the end of the three weeks suckling period, two of the young, all of which were undersized, were given a mixed diet and the other two the 18 per cent. protein modification of Nestlè's Food. Neither group attained the weight proper for their age, but the rate of growth was normal and the experimental mixture quite as efficacious in promoting growth as the general diet. To confirm this conclusion, the young mice on the experimental food were given a mixed diet for one week. No appreciable change in the rate of growth resulted. (See Charts 27 and 28.)

More recently three young mice were given this food from the day they were weaned (twenty-first day after birth). Their growth was much more rapid than that of the controls on mixed food and quite as rapid as that of three others of the same litter which were fed the corresponding modification of Horlick's Malted Milk. (See the two upper lines in Chart 27.)

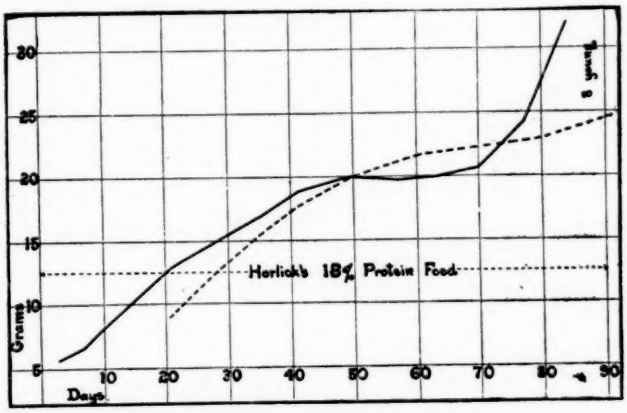

Chart 14.—Mouse 142 ♀, showing normal growth and a normal pregnancy on the 18 per cent. protein modification of Horlick's Malted Milk.

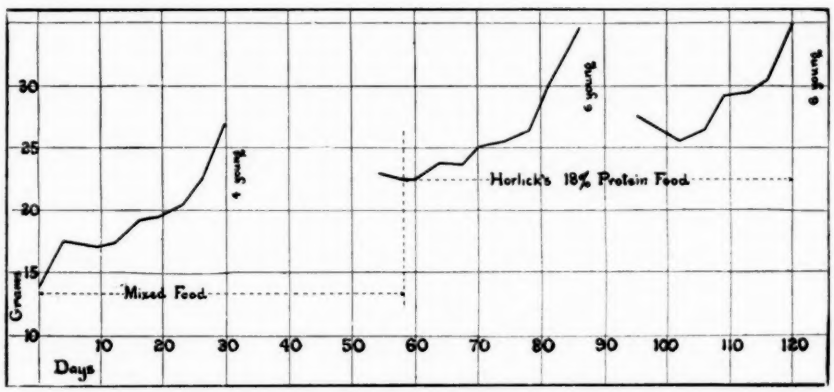

Chart 15.—Mouse 22 ♀, showing three normal pregnancies, one with a mixed diet and two with a diet of the 18 per cent. protein modification of Horlick's Malted Milk. (See Chart 16 in which similar curves are given for a mouse on a mixed diet.)

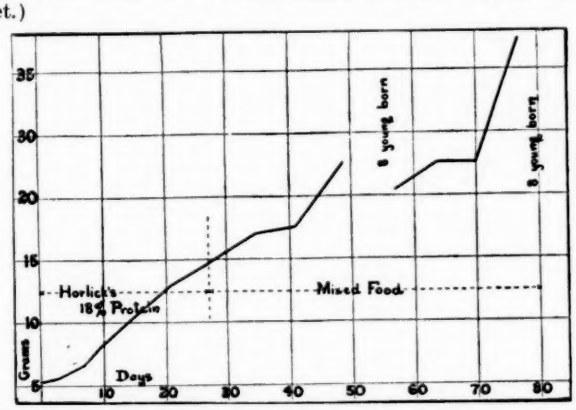

Chart 16.—Mouse 146 ♀, showing two normal pregnancy curves in a mouse on a mixed diet. (See Chart 15.)

Mellin's Food; 18 Per Cent. Protein Modification.—This mixture is especially interesting because it is so closely similar in gross composition to the corresponding modification of Horlick's food and because, like the latter, it contains a large part of its carbohydrate in the form of maltose. One mouse (No. 27, Chart 21), subsisted on this food exclusively for seventy days, weighing at the end of that time slightly more than at the beginning; but during the period two litters were prematurely born, and of all the young, only one lived long enough to be seen alive by the experimenters. Most of them may have been born dead. That the food was responsible seems probable from the fact that on mixed food, this mouse had borne and successfully suckled a litter of eight during the six weeks immediately preceding the experimental period.

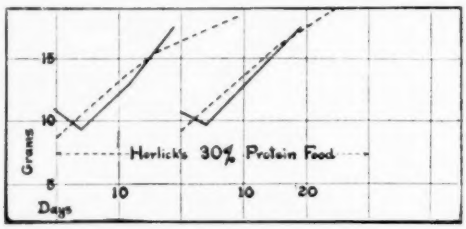

Chart 17.—Mouse 160 ♂ and Mouse 161 ♀, showing normal growth for three weeks on the 30 per cent. protein modification of Horlick's Malted Milk.

This mixture, then, seemed to allow maintenance, but we had no evidence that growth or proper reproduction was possible on it. In the hope of throwing more light on the explanation for this behavior the modification whose composition is given in Column IV under (Mellin's) Food C in the table was given to two mice which had lost approximately one-third of their weight on a diet of unmodified Mellin's Food. The recovery of lost weight was as rapid as on a general mixed diet. (Charts 18 and 19). This remarkable improvement resulting from the addition of only 5 per cent. of casein to Mellin's Food suggests that the proteins of the latter are in some sense inadequate to supply all the nitrogen

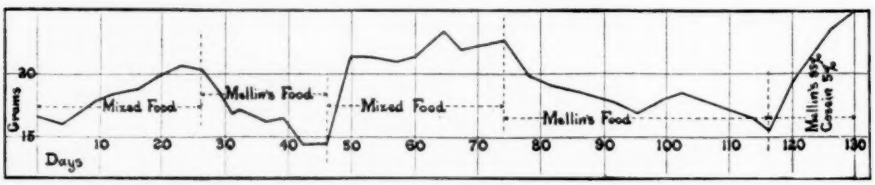

Chart 18.—Mouse 24 ♀, showing loss of weight on unmodified Mellin's Food during two periods and recovery the first time on a mixed diet and the second time on a mixture of 95 per cent. Mellin's Food and 5 per cent. casein.

requirements of the body, for in amount these proteins are equal to those in the satisfactory Nestlè's Food (11 per cent. in each; see columns marked 1 under each food in the table). Even Modification IV, however, allowed only repair. When the original weight was reached, there was no further gain.

Since the above was written, the modification of Mellin's Food last described was fed to three young mice for five weeks. They maintained their weight, but almost no growth occurred.

Eskay's Food: 18 Per Cent. Protein Modification.—Eskay's Albuminized Food contains comparatively so little protein and salts that to make a ration comparable with the corresponding modifications of the other foods, it was necessary to use only 67 per cent. of the food and to add 33 per cent. of milk con-

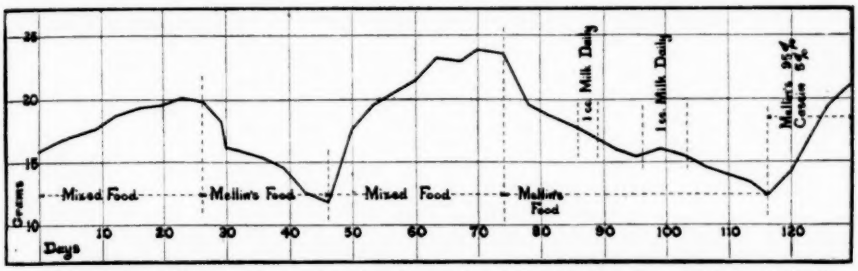

Chart 19.—Mouse 11 ♂, showing loss of weight during two periods of feeding with Mellin's Food and recovery when for this ration was substituted either mixed food or a mixture of 95 per cent. Mellin's Food and 5 per cent. casein. The addition of 1 c.c. milk daily to Mellin's Food had little effect.

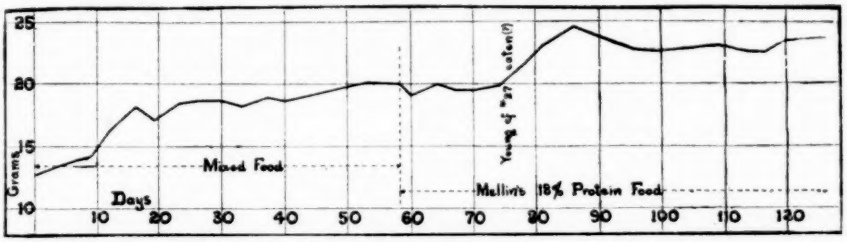

Chart 20.—Mouse 50 ♂, showing maintenance for sixty-eight days on the 18 per cent. protein modification of Mellin's Food. There is a possibility that this animal ate some of the young born in the cage on the seventy-sixth day. If so the effect was greater and more lasting than that from 1 c.c. milk daily for four days. (See Chart 19).

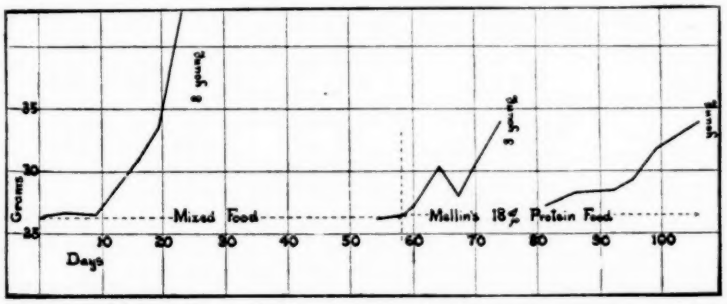

Chart 21.—Mouse 27 ♀, showing a normal pregnancy curve on mixed food and two abortions on the 18 per cent. protein modification of Mellin's Food.

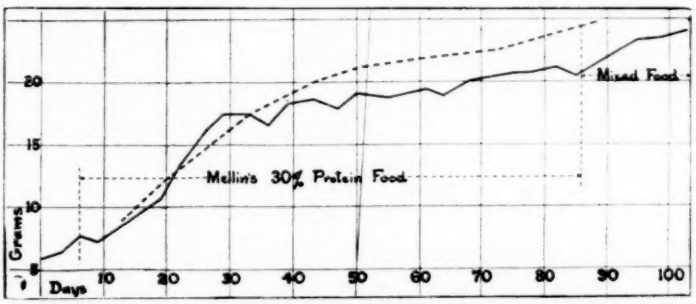

Chart 22.—Mouse 93 ♀, showing approximately normal growth on the thirty per cent. protein modification of Mellin's Food.

stituents (purified casein and protein-free milk). Even then two of the three animals to which the mixture was given lost weight rapidly and died within twelve and fourteen days, respectively. The third mouse, after an equally rapid decline for twelve days, rallied and during the following sixty-five days slowly regained the lost weight. No further gain took place, however; there was only repair; no real growth. (Charts 4, 5 and 6.) Here again the difficulty is not due to the gross composition of the food, which in this respect closely resembles the most successful mixtures. According to Boyden, 41 per cent. of the carbohydrates of Eskay's Food are in the form of raw starch. Are the animals unable to deal with this and is the one recovery due to an acquired or increased diastatic

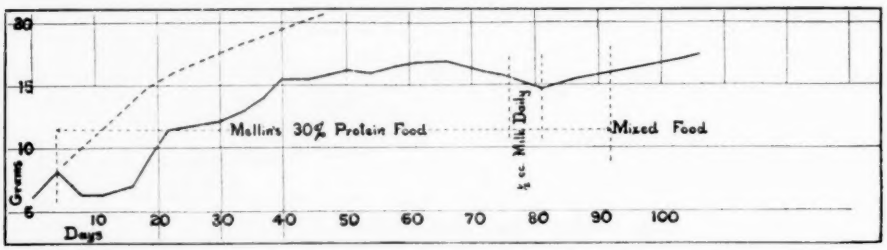

Chart 23.—Mouse 81 ♂, showing growth on the 30 per cent. protein modification of Mellin's Food. One-half c.c. milk daily seemed to check the decline that began after sixty-two days of growth.

power that came with continued eating of the food? It was not a question of the age of the animal for Mouse 7, an adult mouse, showed no more ability to assimilate the food than did Mouse 113, an immature animal. Four of the five cases of diarrhea noticed among the forty-two experimental animals occurred in mice living on Eskay's Food mixtures. As among babies, this condition is often an indication of an excess of carbohydrate in the diet, so here it may indicate unassimilable carbohydrate. Experiments are in progress which it is hoped

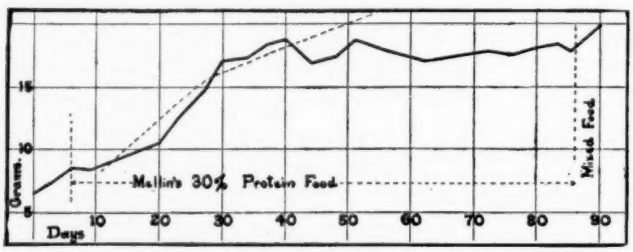

Chart 24.—Mouse 90 ♂, showing growth for thirty-four days on the 30 per cent. protein modification of Mellin's Food and maintenance for forty-seven subsequent days during which the controls were growing steadily.

will settle this point. So far, it has not proved possible to keep mice in health on this modification of Eskay's Food even when the food has been boiled as directed by the manufacturer (but without milk).

Series III—30 Per Cent. Protein Modifications

As is indicated in Column 3 under each food in the table, so much casein and protein-free milk had to be added in order to bring the protein and salt content of the foods up to 30 per cent. and 6 per cent., respectively, that only 25

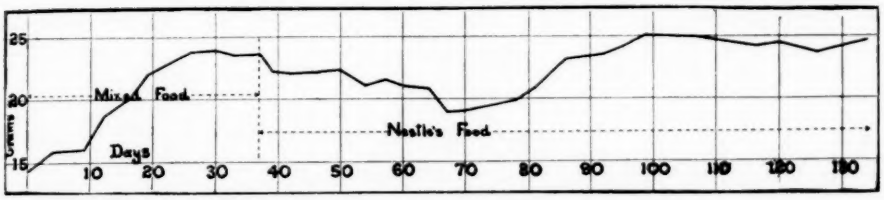

Chart 25.—Mouse 29 ♂, showing maintenance for ninety-one days on unmodified Nestlè's Food.

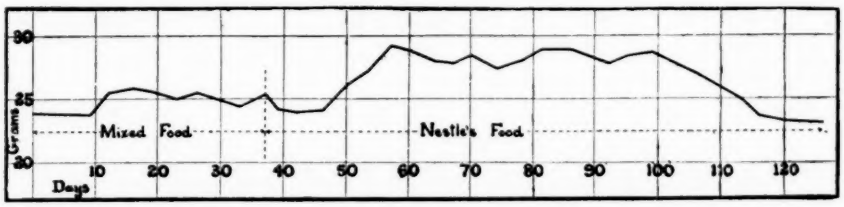

Chart 26.—Mouse 39 ♂, showing maintenance for sixty-two days and subsequent decline on unmodified Nestlè's Food.

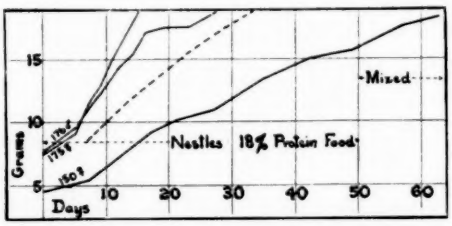

Chart 27.—Mouse 150 ♀ (lower solid line), showing slow growth on the 18 per cent. protein modification of Nestlè's Food. Since a change to a mixed diet had no affect on the rate of growth, the slow gain was probably not due to the food. This was recently confirmed by the rapid growth of Mouse 175 and Mouse 176 on the same Nestlè's Food as shown in the two upper solid lines.

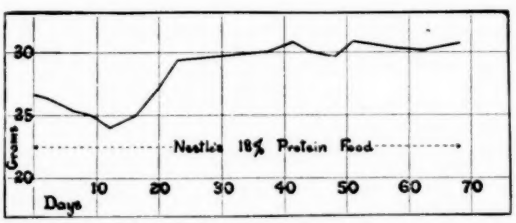

Chart 28.—Mouse 33 ♂, showing maintenance and growth in a mature mouse on a diet of Nestlè's Food modified to contain 18 per cent. protein.

per cent. or less of the original foods could be used, and from 62 to 70 per cent. of the powder as fed consisted of milk constituents. It is not surprising that there was more uniformity in the results of this series of feeding trials than in the preceding ones. In this modification, all four of the foods allowed normal or nearly normal growth for a time, but mice on Mellin's or Eskay's Foods, after three to four weeks of more or less steady gain, generally ceased to grow and in some cases even lost weight. (Charts 7, 9, 22 and 24.) Mice fed the corresponding Nestlè's Mixture grew as rapidly as the controls on a mixed diet. (Charts 19 and 30.) Owing to an accident, the Horlick's 30 per cent. protein mixture could be fed only three weeks, during which time steady growth occurred at a greater rate of gain than that of the controls, but the time was too short to allow safe drawing of conclusions. (Chart 17.)

DISCUSSION OF RESULTS

As criteria for judging a restricted ration, (1) the maintenance of constant body-weight in adult animals for a considerable fraction of their natural life span, (2) the production of normal young, (3) the growth of the young to sexual maturity and maximal weight for the species, and finally (4) the production of a third generation, would

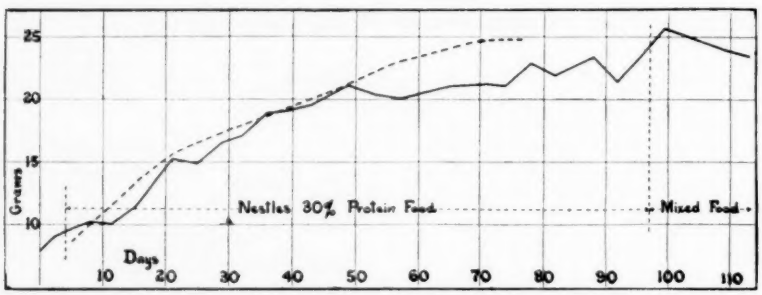

Chart 29.—Mouse 79 ♂, showing growth on the 30 per cent. protein modification of Nestlè's Food.

seem sufficient to prove the ration under investigation a complete food, for the species in question at least. One of the foods containing milk has fulfilled all these conditions; the other (Nestlè's food) the first two and the third so far as a normal rate of growth is concerned; both foods with modifications that involve only quantitative changes in the food constituents. The foods made entirely from cereals (Mellin's and Eskay's foods) seem to be incapable of serving as complete foods for mice under the conditions of these experiments. The inadequacy may be due to (1) the character of the nutrients or (2) the absence of some essential accessory substance such as a vitamine or a growth hormone—provided that we have in our modifications eliminated differences in the total energy value and in the relative proportion of nutrients in the ordinary sense of the word. The difference in energy value between the day's ration of Horlick's food and of Mellin's food is so very small — corresponding to .04 of fat — that it seems unlikely that

it is significant; but the point is now being tested, in view of the fact that the unsuccessful mixtures were of somewhat lower energy value than the successful ones.

ACCESSORY FOOD SUBSTANCES

With these uncertainties it is impossible to settle the question of the presence or absence in the two less satisfactory foods of essential accessory substances, using this term to cover growth hormones, vitamines, etc. Such accessory substances may have been partially responsible for the improved rate of growth that occurred when large quantities of casein and protein-free milk were added to the foods, as in the 30 per cent. protein modifications; but the giving of small quantities of fresh milk as suggested by Hopkins resulted in most cases in no decisive change in the rate of gain or loss of body-weight in progress before this addition to the diet. (Charts 1, 8, 19 and 23.)

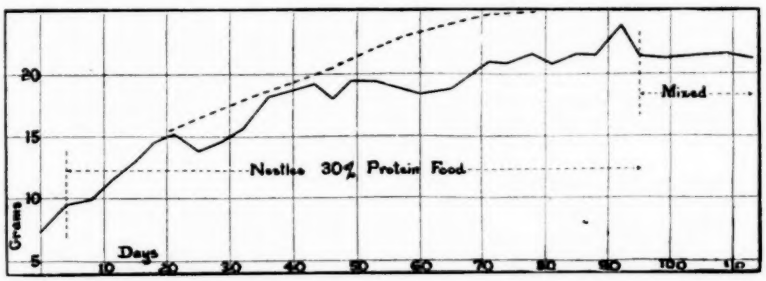

Chart 30.—Mouse 76 ♂, showing growth on the 30 per cent. protein modification of Nestlè's Food.

SUMMARY

Four typical proprietary infant foods were fed to albino mice, both adult and young animals being used. To adapt it to use by mice each food was modified by the addition of purified casein or of the salts of milk (Osborne and Mendel's protein-free milk) or of both.

Two of the four foods seemed to be complete foods (Horlick's malted milk and Nestlè's food), allowing maintenance of constant body-weight, normal reproduction and growth more rapid than normal. With Horlick's malted milk a third generation was produced.

The other two foods studied, Mellin's food and Eskay's albuminized food, did not prove capable of serving as an exclusive ration. One modification of Mellin's food appeared to satisfy the nutritive requirements for maintenance and for repair in two instances but not for growth.

THE PYLORUS, PYLOROSPASM AND ALLIED SPASMS IN INFANTS*

ALFRED F. HESS, M.D.
NEW YORK

In studying the extensive literature of pylorospasm, one is impressed time and again by the striking uncertainty attending its diagnosis and the difficulty of differentiating this condition from organic stenosis and even from simple vomiting. Most writers on the subject have drawn attention to the diagnostic difficulties and have grouped their cases under the title of pylorospasm in a rather tentative spirit, because, all things considered, the symptoms seemed to point most clearly to a spastic condition. Indeed, diagnosis has not always been possible even after post-mortem examination. The problem, however, is essentially a clinical one, as the majority of cases recover and do not reach autopsy. Few require surgical intervention; so that this opportunity for studying the condition is also not frequently afforded.

As the result of clinical experience some observers have come to the conclusion that there is no pathognomonic sign or symptom which serves to differentiate functional from organic pyloric obstruction. On the other hand many have singled out a symptom or symptom-complex which they believe constitutes a significant distinction between the two groups of cases. These various criteria will be fully discussed in the body of this paper. The cases we report have been subjected to a test which was suggested nearly two years ago in a preliminary presentation of this subject[1]— the passage of the duodenal catheter. We shall consider only pylorospasm and not organic obstruction of the pylorus, using as the basis of our report and conclusions some twenty cases of spasm which have been tested by means of the catheter and also studied from a clinical viewpoint. Under this heading of pylorospasm we have assembled cases which, in spite of obstruction at the pylorus, have permitted the passage of the catheter beyond the pyloric sphincter. It is assumed that whenever it is possible to insert a No. 15 (F.) catheter into the duodenum, organic stenosis is so slight that it can be disregarded from a clinical point of view. We do not mean to state that stenosis may not exist in such cases, but that, if we are to differentiate clinically between organic and functional conditions, the obstruc-

* Submitted for publication Dec. 23, 1913.

1. Hess, A. F.: Am. Jour. Dis. Child., March, 1912.

tion in these instances must be regarded as spastic in nature and the disturbance as functional.

MEASURING THE NORMAL PYLORUS

In a consideration of the question of pyloric obstruction in infants, one of the first steps should be to ascertain, if possible, the normal functional diameter of the pylorus and the physiological variations from this form which occur at this early period of life. This question has been considered by many investigators. Various ingenious tests for measuring the lumen of the pylorus have been devised, especially by Pfaundler,[2] who has pursued this study with particular care. However, it must be borne in mind that all tests have been based on post-mortem examination and that they furnish purely anatomical measurements which cannot be regarded as functional. We have confined ourselves to tests *in vivo*. It may be recalled that in the preliminary study on infants a month or more of age it was found that a No. 15 (F.) catheter could be readily inserted past the pylorus. We have made further tests in this direction, using a series of catheters of increasing size and of the following circumferences: 15, 17, 18, 20, 22, 24 mm. We were absolutely unable to pass the No. 24 (F.); the No. 22 was passed occasionally in infants 6 months of age or over. On infants 2 to 3 months of age catheters measuring 15, 17 or 18 mm. could frequently be inserted beyond the pylorus, and exceptionally one having a circumference of 20 mm. From these tests it would seem that *the functional circumference of the pylorus at this age is about 18 mm. and the functional diameter about 6 mm.* It is realized that these figures are not mathematically correct; indeed, it is evidently impossible to lay down hard and fast measurements for the lumen of any sphincter. The sphincteric aperture was found to vary and not to observe a strict parallelism with the size of the infant. In the breast-fed infant, or in any robust, well-nourished baby, the pylorus as a rule has a greater tonicity under normal conditions than in the poorly nourished infant, where the general musculature is relaxed. However, these variations are not as great as, at the first glance, they would seem; the difference between permeability for a No. 18 and a No. 21 catheter is less than 1 mm. in diameter. It is of course perfectly possible that the pyloric musculature, which is highly elastic, is capable of further stretching or dilatation under exceptional pressure, and that, as in the case of Ibrahim,[3] the pylorus, by assuming a slit-like form, may allow the passage of a coin or other object of a greater diameter. Such variations are inherent in

2. Pfaundler, M.: Bibliotheca Medica, 1898, Part 5.

3. Ibrahim, J.: Jahrb. f. Kinderh., 1913, lxxvii, Part 2, p. 199.

functional tests, which, we may repeat, by their very nature cannot be rigid or arbitrary.

Just a word as to the size of the pylorus of infants during the first week of life. An examination of about 100 unselected cases was carried out, including large and small infants, male and female, first-born and those of multipara, cases in which delivery had been easy as well as those in which instrumentation had been necessary. The caliber of the pylorus was found very constant — a No. 14 (F.) catheter could be passed without any difficulty, exceptionally a No. 15; the latter met with evident obstruction and was very tightly gripped by the sphincter. *The functional circumference of the pylorus in the new-born is therefore about 14 mm. and the functional diameter about 4.75 mm.*[4]

CONGENITAL PYLOROSPASM

So much for the normal. There are some abnormal cases in the new-born which would seem of especial interest in connection with a study of pylorospasm. This view can perhaps be brought out most clearly by detailing clinical notes of some illustrative cases:

Kaplan (male); first child; 7 pounds; 7¼ hours old. Not fed. Feb. 27, 1912.

2:34.—No. 14 catheter passed into stomach. Pharyngeal reflex (Ph.R.) increased. Marked Congo +++ fluid in stomach and throughout ten-minute test. Profuse secretion of saliva. Catheter sucked greedily.

2:50.—Catheter passed again. Ph.R. increased. Duodenum entered easily, marked grip. On entering stomach less grip, marked pharyngeal grip. Baby quiet.

3:06.—Reintroduced. Ph.R. increased. Some obstruction at pylorus, cardia and pharynx; most marked at cardia. Infant sneezed as catheter passed pylorus.

3:22.—Retraction into stomach, where Congo ++ fluid was found. Grip on catheter less when in stomach; still less when in esophagus.

Epicrisis: Marked pharyngeal, cardiac and pyloric reflex. Increased secretion of hydrochloric acid.

This case is cited because it illustrates in an unfed, new-born infant various signs that we shall have occasion to refer to later in connection with pylorospasm — the increase in the pharyngeal reflex, the association of this hypersensitiveness with increased reactivity of the cardiac and pyloric sphincters, and the hypersecretion of gastric juice.

---

4. The pylorus of an infant had no occasion normally to dilate to this extent. The "pyloric casts" of milk which can be aspirated from the duodenum (as referred to in an article on casein curds) have a diameter of 1 to 2 mm., and denote the normal diameter which the pyloric canal assumes during the passage of milk from the stomach into the duodenum. Pfaundler found the inner circumference of the pylorus, measured after relaxation of the musculature, to be as follows: At birth 2.0 cm.; at 3 months, 2.4 cm.; at 6 months, 2.7 cm.; at 9 months, 3.0 cm.; at 1 year, 3.3 cm. A comparison shows, therefore, that these circumferences are far larger on the cadaver than during life.

A second case brings out some of these phenomena still more clearly. It is reported in abstract as there were nine separate tests:

Baby Blum; 24 hours old. Weight 7 pounds 6 ounces.

Test 1. Jan. 23, 1912. Pharynx reflex (Ph.R.) negative. Congo ++ fluid in stomach.
Test 2. Jan. 24, 1912. Ph.R. increased.
Test 3. Jan. 25, 1912. Ph.R. increased on three tests.
Test 4. Jan. 26, 1912. Ph.R. increased.
Test 5. Jan. 27, 1912. Ph.R. increased.
Test 6. Jan. 29, 1912. Ph.R. negative. Pylorospasm. Could not enter duodenum without putting water into stomach (accessory method). Marked grip on catheter. Alkaline bile aspirated.
Test 7. Jan. 30, 1912. Ph.R. negative. Indefinite whether entered duodenum.
Test 8. Jan. 31, 1912. Ph.R. increased. Seemed to catch in pylorus. In a second test, carried out with the aid of accessory method, catheter was felt suddenly to pass the pylorus.
Test 9. Feb. 1, 1912. Ph.R. increased. Could not enter pylorus without aid of accessory method.

Epicrisis: Repeated pharyngospasm and pylorospasm.

In this instance the spastic condition was almost constant, either as pharyngospasm or as pylorospasm. This same condition was met with in another case (No. 40) where nine tests were also made. In seven an increase of the pharyngeal reflex was found, at times associated with pylorospasm.

The spastic manifestations, however, are not always so constant. The following case illustrates this variability:

Polack. Weight 7 pounds 7½ ounces. Fifteen hours old. Unfed.

Test 1. Dec. 19, 1911.
4:13. No. 14 catheter passed. Ph.R. normal. 0.5 c.c. Congo +++ fluid.
4:15. Catheter reintroduced. Cardiospasm; definite obstruction 13 cm. from gums. Difficulty in passing pylorus, crying, no gagging.
Test 2. Dec. 26, 1911.
4:18. Ph.R. increased. 1½ oz. slightly acid, Congo negative milk in stomach. No cardiospasm.
4:25. Reinserted, easily entered duodenum. 2 c.c. yellow viscid fluid; also pyloric casts.
4:40. Retracted; no free HCl in stomach.
Test 3. Dec. 27, 1911.
3:25. Ph.R. increased. 1½ oz. Congo negative fluid in stomach.
3:32. Easily entered duodenum. Bile. Pyloric casts. No free HCl in stomach.

Epicrisis: In a test soon after birth marked cardiospasm and some pylorospasm was encountered. In two tests carried out a few days later these sphincters were found normal, but pharyngospasm was met with.

In addition to the cases cited or referred to above, other new-born infants were found to have an abnormal irritability of the pharynx, cardia or pylorus; eight cases in all, among one hundred infants tested during the first few days of life. We shall not at this time enter on the interesting subject of the interrelationship of these various forms

of spasm, as this phase of the subject will be considered at length when treating of spasm in later infancy. We wish merely to call attention to the presence of these spastic conditions at and soon after birth, at a period when post-natal influences have not played a part, and when all abnormal conditions must necessarily be considered congenital and perhaps hereditary. *These, then, are true cases of congenital pharyngospasm, cardiospasm and pylorospasm.* Similar phenomena may be met with during the first days of life, excited by post-natal intercurrent factors, by gastric disturbance or dyspepsia. One case of this nature was encountered on which seven tests were made (cited below in abstract), three previous to the alimentary disturbance and four subsequent thereto. Before the dyspepsia developed the infant seemed normal with the exception that pharyngospasm was encountered in one of three tests. Soon after the dyspepsia developed (as evinced by vomiting, fermented gastric contents and abnormal stools), the pharyngospasm increased and became constant, and pylorospasm was demonstrated by the duodenal catheter:

Schneider, 5½ hours old. Unfed. Weight 7 pounds 12 ounces.

Test 1. Jan. 22, 1912. Ph.R. increased. Salivation. Small amount of HCl.
Test 2. Jan. 23, 1912. Ph.R. normal. Catheter introduced three times.
Test 3. Jan. 24, 1912. Ph.R. normal. Entered duodenum. Pyloric cough.
Test 4. Jan. 25, 1912. Ph.R. normal. Stools greenish, with curds. Sour odor to stomach contents.
Test 5. Jan. 26, 1912. Ph.R. increased. Stomach contents curdled, acid odor. Stools green. Difficulty in passing pylorus. Could not enter in spite of many attempts. *Pylorospasm.* Accessory method used; felt sudden relaxation, entered duodenum. Catheter held, not ejected.
Test 5. Jan. 26, 1912. Vomits occasionally. Ph.R. increased. Catheter held tight when introduced into stomach. Difficulty in entering duodenum; succeeded only by means of accessory method. Infant fretted until catheter passed pylorus.
Test 7. Jan. 29, 1912. Occasional vomitings; curds in stool; slight prolapse of rectum. Ph.R. increased. Pylorospasm; accessory method necessary to pass pylorus.

Epicrisis: First tests normal except for pharyngospasm on one occasion. Dyspepsia followed by pylorospasm as well as pharyngospasm.

What is the later clinical history of these infants who manifest pharyngospasm, cardiospasm or pylorospasm almost from the hour of their birth; how do they pass through the first months of life, the critical period from the standpoint of pylorospasm? We have had an opportunity to follow five of these cases after they have been discharged from the maternity hospital. Owing to the usual difficulties which surround follow-up work in a large city, the surveillance in all cases was not as long as is desirable. All the infants were nursed throughout the period of observation. Three (Glaisier, Blum, Schneider) remained

entirely well throughout the first three months of life and thrived satisfactorily; one mother, however, reported that for a considerable period the baby (Schneider) vomited repeatedly. Little stress is laid on this indefinite report. The course was not so smooth in the progress of two of the infants: one (Plevner) began to vomit at irregular intervals when 1 month of age, the vomiting being projectile in character. This continued until he was 3 months of age. He was fairly nourished, not emaciated. Probably a mild form of pylorospasm existed, to which we shall refer below. The other infant (Fleischman), to whom we have not previously referred, showed cardiospasm, pylorospasm and gastric hypersecretion (6 c.c. Congo + + + fluid, free HCl 19, combined HCl 37), when examined at birth, on Nov. 18, 1911, before being fed. He began to vomit when about 2 months of age, irregularly, but daily. When visited on January 31, examination showed a fairly nourished infant, gastric area distended, no visible peristalsis. Propulsive vomiting seen. Catheter readily passed. On February 2, at the time of the second visit, some difficulty was experienced in passing the catheter. The vomiting was about the same, but became less marked and by February 16 had ceased. The baby continued to improve; it was last visited April 13.

It would seem that in some new-born infants there is a congenital tendency to spasm of the sphincters to which we have referred, and that clinically this condition may remain entirely latent or may manifest itself in a slight disturbance, such as occasional projectile vomiting, or may become evident following superimposed dyspepsia or other exciting cause. This then may be properly termed congenital pylorospasm. As we have seen, it is frequently latent, and associated vicariously with pharyngospasm and cardiospasm.

Before leaving this subject of spasm in the new-born, we wish to call attention to the fact that the tonus of the pyloric sphincter may be abnormal not only in a positive, but also in a negative sense; that instead of spasm the duodenal catheter may reveal a relaxed or atonic condition. This was noted in two instances. One has been referred to in quite another connection in a study of icterus neonatorum.[5] The case (Seep, Case 16) was one of congenital family jaundice. The infant was saffron-colored and toxic. There was a marked insufficiency of the pyloric sphincter. Bile was found in the stomach in five of the six tests, and there was vomiting of bile. The catheter could be passed in and out of the duodenum without the least obstruction. Associated with the relaxation in this instance there was diminution of the pharyngeal reflex. Marked relaxation of the pylorus was encountered in

5. Hess, A. F.: Am. Jour. Dis. Child., May, 1912.

another case (Sapperstein) in which there was intense jaundice. It has been shown that these cases of jaundice are associated with a profuse excretion of bile into the duodenum; indeed, generally with a welling forth of the bile into the stomach.[6] The abnormal patency of the sphincter would seem to result from this increased biliary secretion, to be based on the persistent alkalinity of the upper duodenum, which is acknowledged to hold the latch-string of the pylorus.

In these cases we were confronted with an atonic state of the sphincter, leading to *pyloric insufficiency* as the result of the local inhibition of the pyloric closing reflex. Another instance showed that we may encounter the same functional muscular insufficiency in the course of the examination of an infant otherwise apparently normal:

Qualler, 3 hours old. Weight 6½ pounds. Pale; not strong.

Test 1. Feb. 1, 1912. Ph.R. diminished. Marked saliva, free HCl in stomach.

Test 2. Feb. 2, 1912. Ph.R. diminished. Free HCl in stomach. Inserted catheter three times into duodenum with the greatest ease.

Test 3. Feb. 3, 1912. Ph.R. diminished. Catheter introduced into duodenum without meeting the least resistance.

Epicrisis: Diminished reflex of the pharynx and of the pylorus.

II.

Following these preliminary remarks concerning pylorospasm in the new-born, let us turn to a consideration of various points of interest encountered in the course of passing the duodenal catheter on a large number of older infants, and more especially in cases of pylorospasm. It seems preferable, in order to bring out saliently the various phenomena which we wish to discuss, to consider the subject from the standpoint of various isolated symptoms, rather than as a clinical entity. We shall therefore take up the following pathological symptoms, always bearing in mind that they rarely occur singly and that the subdivisions are purely artificial: pharyngospasm, cardiospasm, visible peristalsis, palpable tumor, gastric and duodenal hypersecretion, singultus, pylorospasm and allied spasms.

Many of the cases were seen at the Post-Graduate Hospital on the service of Dr. Henry D. Chapin, to whom I take pleasure in expressing appreciation for the privileges accorded me.

PHARYNGOSPASM

First, as to the occurrence of pharyngospasm, described in this connection by Finkelstein. One cannot pass a catheter on many normal infants, and especially on infants suffering from spasm of the pylorus,

6. Hess, A. F.: Am. Jour. Dis. Child., October, 1913.

without having the attention drawn to the intimate relationship between the reflex of the pharynx and that of the pylorus. Frequently when the tip of the catheter engages the pyloric sphincter the infant begins to gag, and at each repetition of the procedure will react in the same way. We shall not attempt to give a physiological explanation of this connection. However, it is undoubtedly true that when the pyloric reflex is increased, the pharyngeal reflex generally is also increased, and that there is also at times a connection between the reflex at the cardia and the reflex at the pharynx. It will be remembered that we cited an instance in the new-born in which there was an insufficiency of the pyloric sphincter and also a lack of reactivity on the part of the pharynx (Seep case).

Associated with spasm of the pharynx muscles there may be gastric hypersecretion. In one instance, by instilling a small amount of normal hydrochloric acid into the duodenum and thus causing a contraction of the pylorus, a spasm of the pharynx was brought about. In the only instance in which hydrochloric acid was found lacking at birth[8] the pharyngeal reflex was markedly diminished.

Pharyngospasm usually manifests itself as gagging, the well-known pharyngeal protective reflex which is encountered when one attempts to introduce any object past the pharynx. This reflex activity may vary somewhat in type. It may be merely a tonic spasm, without any gagging, the pharynx muscles contracting tonically and preventing the passage of the catheter. On the other hand, it may be accompanied by marked retching and gagging, persisting as long as the catheter is kept in place. This was met with but once in the new-born (Glaisier case). It is exceptional in infants, although common in adults. Or the pharyngospasm may be incited only after a second or a third passage of the catheter. These various manifestations are of interest but of no clinical importance. We wish, however, particularly to emphasize not only that there is a close relationship between pharyngospasm and pylorospasm, but that the pharyngospasm may be an important factor in these cases. In several instances it has resulted in vomiting immediately on the baby's being put to the breast or given the bottle. In one of these cases, in which bismuth was added to the milk, the Roentgen-ray showed that food had not entered the esophagus. The grip of the pharyngeal muscles in these cases is frequently so tight that the fluid cannot be passed through the catheter without considerable force; in one instance (Stark) a column of 30 cm. of water was insufficient to overcome the resistance. At times fluid cannot be aspirated until the catheter is withdrawn into the mouth. In citing cases we have referred to this phenomenon as the "pharyngeal retraction test." It is met with occa-

sionally in infants who appear otherwise quite normal, and therefore cannot be considered indicative of pylorospasm.[7]

CARDIOSPASM

In cases of spasm of the pylorus, associated or not associated with pharyngospasm, we may encounter cardiospasm. The frequency of cardiospasm under these conditions was emphasized in our preliminary article, and subsequent experience has served only to strengthen our opinion as to the frequency and importance of this symptom. Our remarks are based on the observation of eight cases in which there was cardiospasm. A few words seem in place regarding the physiology and the pathological physiology of this condition. V. Mikulicz observed in man that on the esophageal side such slight pressure as from 2 to 7 cm. of water was sufficient to drive air or water into the stomach. As a rule, the necessary pressure was less than that of a column of fluid which would fill the thoracic esophagus. The normal tonus of the cardia would therefore seem to be slight. As regards the control of the cardia, Cannon[8] has suggested that "just as acid beyond the pylorus keeps the pylorus closed, so, likewise, acid in the stomach (beyond the cardia) may keep the cardia closed." Reviewing our tests from this point of view, we find that although some cases of cardiospasm were associated with gastric hyperacidity (cf. Udo case below), others undoubtedly were associated with a normal or subnormal degree of hydrochloric acid, as was demonstrated by a marked case of cardiospasm associated with pylorospasm:

David Ross, 7 weeks old. Weight 6 pounds 14 ounces. Breast fed for four weeks; since then ¼ milk, ¾ water. Has vomited and been constipated. In hospital two weeks, fed every three hours 4 ounces of a mixture of half milk, half water, with the addition of milk sugar. Has been vomiting almost every feeding until the last twenty-four hours. Fed 2½ hours ago. Pale, nervous child. Pupils constantly dilating or contracting.

Nov. 13, 1911. Ordinary stomach tube passed to empty stomach. Difficulty in passing beyond 15 cm. (cardia). Stomach absolutely empty. "Duodenal ball-tube" then used, but could not be inserted beyond cardia, even after giving water (accessory method). Duodenal catheter passed. Obstruction again at cardia. Duodenum entered with some difficulty; 5 c.c. of thick, mucoid, bile-tinged fluid obtained in thirty-minute test. No free HCl in stomach.

Epicrisis: Marked cardiospasm unaccompanied by gastric hypersecretion. Pylorospasm slight or absent.

If even a soft catheter is introduced rapidly into the stomach, a mild grade of cardiospasm will be overcome and may be overlooked,

7. In one case the "duodenal ball-tube" encountered extraordinary resistance at about the level of the manubrium on attempting to withdraw it from the esophagus. It is difficult to explain this marked constriction. Possibly it was due to the pressure of an enlarged thymus, and the ball-tube may be of value in the diagnosis of this condition.

8. Cannon, W. B.: Mechanical Factors of Digestion, 1911, p. 39.

especially if attention is not paid when the level of the cardia is reached. When, however, a soft rubber tube with a metal ball at the end is introduced (the "duodenal ball-tube"),[9] mild grades of this pathological condition are frequently encountered. The tonicity of the cardia varies greatly within physiological limits.[10] However, if the sphincter grips the catheter so tight that we are unable to aspirate fluid until it is retracted into the esophagus (cardiac retraction test), we may consider that there is abnormal spasticity present, as, for example, in the following case, which is illustrative from this point of view, and also for the association of spasm of the pharynx, of the cardia, and gastric hypersecretion (cf. case A. W., reported in the previous paper):[9]

Annie Udo, 3 months, 7 pounds. Atrophic.

Test 1. March 15, 1912. History of vomiting. Fed at 3 o'clock.

3:30. Catheter passed. Ph.R. increased. Also spasm at cardia. Congo ++ fluid in stomach. Entered pylorus. Reintroduced. Cardiospasm again. Congo +++ in stomach. Entered pylorus; "cardiac retraction test." Fluid could not be aspirated until entered esophagus. Then 2 c.c. of bile obtained.

3:55. More cardiospasm. Congo ++ fluid in stomach.

Epicrisis: Pharyngospasm, cardiospasm, gastric hypersecretion.

Test 2. March 18, 1912. Fed at 12 o'clock.

1:10 Ph.R. increased and Congo ++ in stomach. Reintroduced. Congo +++ fluid.

1:15. Water could not be made to pass through catheter by gravity into stomach. All fluid Congo +++ markedly increased in quantity.

Test 3. March 20, 1912. Some retraction of head; rigidity of legs. Fed at 12 o'clock. Thirty-five-minute test showed pharyngospasm, cardiospasm and gastric hypersecretion, as in the two previous tests. Pylorus entered twice and bile obtained; but the condition of the sphincter could not be judged on account of the spasm of the cardia.

The obstruction at the cardia in this case was moderate. However, it may reach such a degree that we are unable to enter the stomach without passing a stylet through the catheter. We had this experience in two cases (McGee and Horowitz), one of which we shall cite:

James McGee, 3 months, 6½ pounds. Has vomited large quantities of blood. Fed three hours previous to test, 4 ounces half-milk with sugar, alternating with breast feeding.

Catheter passed. Ph.R. normal. Could not enter stomach until inserted a ureteral stylet through catheter. Stomach empty. No acid. Entered duodenum easily. Bile alkaline, ++, but could not aspirate it until retracted catheter past cardia.

Epicrisis: Marked cardiospasm (stylet). No pylorospasm found. Probably ulcer of the duodenum or stomach.

---

9. Hess, A. F.: Am. Jour. Dis. Child., May, 1911.

10. This can be ascertained by attaching a small rubber balloon to the end of the catheter, constituting what may be termed a "blind-dilatable-catheter." This can be introduced into the stomach, inflated to various degrees and then withdrawn, in this way measuring the maximal diameter of the cardia. By this means it will be noted that there is a marked variation in the size of the balloon which can be pulled through the cardia in different cases.

In both these cases, as well as in the Ross baby mentioned above, the stomach was found absolutely empty of food although the infant had been fed shortly before passing the catheter. This is typical of what may be termed "cardiospastic vomiting." It is such cases which have led to some of the enthusiastic reports concerning the value of gavage in pylorospasm.

GASTRIC HYPERKINESIS

(GASTRIC DISTENTION, VISIBLE PERISTALSIS, PERISTALTIC TUMOR)

We now come to the consideration of one of the most interesting and striking clinical signs of pylorospasm, one which until lately was believed to be of great significance in the differential diagnosis of this condition. We refer to the peristaltic waves which are frequently seen rolling over the gastric area from beneath the free border of the ribs on the left, to somewhat beyond the median line on the right, and to other gastric manifestations of hyperkinesis. The question at point has been whether visible peristalsis could occur when merely a spasm of the pylorus existed, or whether an organic obstruction was necessary for its production. Of late numerous writers have shown convincingly that this sign may be present in marked intensity where there is no organic stenosis. We shall therefore not discuss this question, regarding it as definitely settled, but shall turn our attention to this phenomenon in cases where the catheter was successfully passed into the duodenum, and where, therefore, obstructive hypertrophy of the pylorus can be ruled out. The question thus resolves itself, not as to whether organic stenosis must be present, but whether even a functional obstruction is essential to visible peristalsis.

This proposition can be answered decidedly in the negative. Upon several occasions we have seen classical peristalsis where there was neither the clinical history nor the clinical picture of spasm, and where repeated testing by means of the catheter revealed an unobstructed path from the stomach into the duodenum. We shall cite one case, selecting an infant which had been under close observation for some weeks before symptoms developed and which was under our care for some months thereafter:

Ephraim K. Admitted to the Hebrew Infant Asylum when 2½ weeks old. Five weeks old, 7¾ pounds. Grippe, followed by dyspepsia; vomiting. Casein masses in the stool. Visible peristalsis; hour-glass contraction of the stomach.

Test 1. March 5, 1912. Five c.c. of acid Congo + milk evacuated from stomach. Peristalsis ceased, although gastric area remained distended. Catheter in stomach did not cause peristalsis. Easily entered duodenum. Bile obtained.

Test 2. March 6, 1912. Alimentary intoxication. Child to be starved for twelve hours. Has not vomited. Observed two hours after feeding; no peristalsis. One ounce of water put into stomach. Immediately peristaltic waves, peristaltic tumor, and hour-glass contraction became visible. Forcible vomiting of the water alongside of catheter before it had entered the stomach. Peristalsis seemed to

be started by swallowing the fluid. Duodenum easily entered. Upon second attempt some cardiospasm. No peristaltic tumor when catheter was in stomach, in duodenum, or held in esophagus for a few minutes.

Test 3. March 8, 1912. Visible peristalsis, hour-glass formation. Some cardiospasm on passing catheter. No pylorospasm (duodenum easily entered).

Test 4. March 10, 1912. Conditions the same. Only occasional vomiting after middle of March. Peristaltic tumor was noted from time to time, even during convalescence. Infant was under observation until June 26.

Similar phenomena accompanying other cases of alimentary intoxication have been encountered. Quite recently the same signs were seen in an infant 1 year of age. However, it is not necessary that the alimentary disturbance be marked. A mild dyspepsia suffices at times to bring about visible gastric peristalsis. This is especially true if the infant is emaciated. On Aug. 21, 1913, an infant (Gordon) was admitted to the hospital suffering from mild dyspepsia. The abdominal wall was so thin that not only could peristaltic waves and a peristaltic tumor be seen, but even the slate-colored liver could be made out. There was, however, no obstruction at the pylorus. We mention this case of marked emaciation in order to advance this phase of the question for consideration. There can be no doubt that a thin parietal wall enables peristalsis to be more readily seen, but it is certainly a factor of marked secondary significance. We may encounter emaciation plus pylorospasm and nevertheless not see peristalsis. For example, Barbas, a 5-months-old infant, weighing less than 6 pounds, had marked propulsive vomiting since birth and gave evidence of pyloric obstruction in the course of the six tests which were carried out, but at no time displayed visible peristalsis in spite of the marked atrophic condition. In this infant the emaciation was marked, pylorospasm present but inconstant, visible peristalsis absent. However, the pylorospasm may be great and nevertheless not give rise to visible peristalsis. We shall cite in abstract a case of this kind, as it is frequently taken for granted that the spasm incites the marked peristalsis, the conception being that the contractions of the stomach become visible in the course of its efforts to overcome the obstruction at its exit:

Dede, 2 months old, weighing 6 pounds 9 ounces, was tested by means of the catheter five times. The obstruction was very great; in fact, at the second test it could not be overcome in spite of repeated efforts. Nevertheless visible peristalsis was at no time present.

From these and similar clinical experiences, combined with the direct testing of the pyloric sphincter by means of the catheter, we are not inclined to lay great stress on visible peristalsis or the peristaltic tumor as a sign of even a functional obstruction of the pylorus. It may be present where there is no obstruction, and may be missing even in atrophic infants, where the catheter cannot make its way past the pylorus into the duodenum.

We were able to approach this aspect of the question from another viewpoint. A catheter similar to the "blind-dilatable-catheter" referred to in connection with cardiospasm was inserted into the duodenum in poorly nourished babies. The balloon was dilated and the catheter retracted, so as to bring about an obstruction of the pylorus.[11] In this way we effected a mechanical obstruction at the pylorus. We can summarize our experiences by stating that although this procedure was carried out on several occasions for long periods, in no instance were we able to incite visible peristalsis.

In this connection we wish to touch on the relation of hydrochloric acid to peristalsis, leaving its more detailed consideration for the paragraph on hypersecretion. In the case of infants some months of age it was not found possible to produce peristaltic waves by instilling small quantities of normal hydrochloric acid into the stomach. However, in the new-born we occasionally succeeded by this means in bringing about ballooning of the stomach and slight peristalsis accompanied by restlessness. This was especially true in the case of nervous, new-born infants (*e. g.*, Rochman, a twin infant 1 day old, who started markedly at the sound of sharp noises. Instillation of 2 c.c. of 0.4 per cent. hydrochloric acid produced marked distention of the gastric area). This ballooning of the stomach is the mildest clinical form of a disturbance of the gastric musculature, of which visible peristalsis, peristaltic tumor (pseudo-tumor), and hour-glass formation are severer manifestations; it is frequently seen days before peristalsis develops, and persists when peristalsis is checked by means of emptying the stomach. Probably all these signs are merely evidences of an intensification of normal peristalsis.

It is difficult to discuss the etiology of gastric hyperkinesis. As we have stated, it is not due to spasm of the pylorus. It is rather a gastrospasm (Thomson), due in part to a hypersensibility of the gastric mucosa, as we must be led to infer from the fact that it is not seen when the stomach is absolutely empty. In this connection, however, it should be noted that where there was marked peristalsis which could be produced at will by introducing even a few cubic centimeters of food, the presence of the catheter in the stomach and within the pylorus did not cause visible peristalsis (*e. g.*, case of Ephraim K.). This was our common experience. Perhaps the presence of the catheter in the pharynx and esophagus may inhibit the muscular activity. This observation seems of practical interest in connection with the passage of the catheter for gavage or for duodenal feeding.

A word as to antiperistalsis. We have not observed it in any case which was proved by means of the catheter to be spastic in nature, so

11. This technic will be entered on in greater detail at another time.

that its consideration does not properly fall within the limitations set for this paper. Its value is greatly circumscribed by the fact that it is so rarely met with. Antiperistalsis, however, is frequently seen by means of the fluoroscope although not visible to ordinary inspection. The study of its occurrence as seen by the Roentgen ray seems far more promising from a diagnostic point of view than the observation of gastric retention, which cannot inform us of the nature of the pyloric obstruction.

Before leaving this subject, we would call attention to the fact that distention and peristaltic tumor of the large intestine are perhaps not infrequently mistaken for gastric manifestations. This has been our experience in several cases in which we have had the opportunity of passing the duodenal catheter. Recently this error was narrowly avoided by being able to palpate the catheter within the stomach above the site of dilatation. Caution should be observed in considering distention as gastric if it appears below a horizontal line drawn midway between the xiphoid and the umbilicus.

PALPABLE TUMOR

The consideration of the occurrence of a palpable tumor will not detain us long, as it was not met within any case of pylorospasm. Nor has it, so far as we know, ever been described where hypertrophy was not found. In this connection, however, a report by Churchman[12] of a palpable spastic tumor of the ileum, encountered at operation, is worthy of attention and reflection. Much depends on the anatomical position of the pylorus in determining whether or not it can be palpated. An hypertrophied pylorus may be rendered intangible by the liver, by the colon, by gastric distention, or by the tonicity of the abdominal muscles; on the other hand, it may be rendered more readily perceptible to the touch as the result of ptosis. We had expected that the normal pylorus would at times be palpable when it contained the catheter. In this, however, we were mistaken. Even in those exceptional cases in which emaciation allowed the palpation of the catheter within the stomach, it could not be followed far beyond the median line; at this point its outline eluded the touch, sinking dorsally beneath the liver.[13]

A "palpable tumor" would seem to signify a perfectly definite anatomical condition. Nevertheless, it is probable that fully as many errors have been made in this particular at operation, and perhaps at autopsy, as in the clinic. It is not always easy to differentiate between a pylorus which is spastic and one which is hypertrophied. The tendency, espe-

12. Churchman, John W.: Jour. Am. Med. Assn.,

13. In one case the catheter was felt within the stomach and also within the descending duodenum lying just external to the right kidney. However, it could not be palpated at the pylorus and within the first portion of the duodenum.

cially for the surgeon, is forthwith to consider the obstruction organic, and there is no doubt that in this way many cases of simple spasm have been reported as organic obstructions.

HYPERSECRETION (GASTRIC, DUODENAL)

It is well known that, associated with pylorospasm, we frequently find a hypersecretion of gastric juice. Indeed, some have attributed the spasm to the abnormal amount of acid or degree of acidity. However, this simple etiological relationship generally does not exist. This is evident from the fact that cases of pylorospasm are not infrequently met with in which there is no increase of hydrochloric acid; this type of case was described in the previous paper, and it has been referred to repeatedly in the reports of others. As further evidence that this interpretation is erroneous, we may add that the amount of hydrochloric acid has been found to furnish no criterion whatsoever as to the facility with which the duodenal catheter can be introduced. Recently we referred to this lack of association in connection with cases of "congenital gastric hypersecretion" of the new-born, and may amplify the statement by adding that in instances of gastric hypersecretion in older infants there may be no obstruction to the catheter and no symptoms significant of pyloric obstruction.

However, we do not mean to infer that hydrochloric acid plays no rôle in the tonicity of the pylorus and the gastric musculature. Test experiments at once convinced us that this view would be fallacious. In the case of a number of infants, varying in age from a few hours to a few months, we introduced into the stomach by means of a tube from 1 to 3 c.c. of 0.4 per cent. hydrochloric acid, and observed the clinical symptoms, the abdominal signs, and the effect as regards the passage of the catheter. The following is a brief summary of the results:

INSTILLATION OF 0.4 PER CENT. HYDROCHLORIC ACID INTO STOMACH

1. SCHRIEDER.—Nine hours old. Unfed. 2.5 c.c. HCl. Marked drum-like distention of stomach; intense cyanosis; repeated vomiting; restlessness; pain, no peristalsis; no hiccough; bowels moved frequently. Passed pylorus.

2. GOLDHABEN.—Seven hours old. 2 c.c. HCl. Rapid respirations; some pain; slight gastric distention; no peristalsis. No duodenal catheterization.

3. MISCHMAR.—Nine hours old. 2 c.c. HCl. Marked contraction of diaphragm forcing out epigastric area. No hiccough.

4. ROCHMAN.—One day old. 2 c.c. HCl. Vomiting, distention of left hypochondrium. No peristalsis.

5. BACONSTEIN.—Two days old. 2 c.c. HCl. Gastric distention; crying; pain. Slight bleeding. Catheterization not attempted.

6. GINSBERG.—Two and three-fourths days old. 2 c.c. HCl. Saliva decreased. Spastic contraction of diaphragm; hiccough; abdominal distention; increased respiration; no peristaltic waves; no pylorospasm.

Test 2.—Five and three-fourths days old. 1.5 c.c. HCl. Hiccoughs in three minutes; no pylorospasm; bile obtained; no vomiting.

7. BABY X.—Five days old. 2 c.c. HCl. Very restful infant. Twice 1 c.c.; no effect. Slight increase in respiration.

8. BERNSTEIN.—Six days old. 1.5 c.c. HCl. Distention of epigastrium; restlessness; pain which ceased on aspirating HCl; no bile; no increased peristalsis. Entered duodenum easily.

9. LEVINE TWIN.—One month old. 2 c.c. HCl. (Baby that has gastric distention and hiccough) 1 c.c. introduced twice. No effect.

10.—LEVINE TWIN.—One month old. 1 c.c. HCl. Some gastric distention; tonic contraction of diaphragm; pain; no hiccough; effect slight.

11. ARGOSE.—Three months old. Normal baby. Intense gagging, caused by putting 3 c.c. HCl into duodenum. Flow of bile.

From these tests it is evident that the introduction of hydrochloric acid, even in a small quantity, such as is normally present in the stomach of the new-born infant, is sufficient to excite motor and reflex phenomena. However, it should be noted that the pylorus seemed to be frequently the least affected part of the musculature of the stomach, the catheter readily passing the sphincter in spite of marked signs and symptoms of gastrospasm. At times vomiting ensued, the gastric area became prominent, there was drum-like distention of the stomach, although no visible peristalsis. In addition, in fact preëminently, there were referred or reflex symptoms, such as gagging (pharyngospasm), hiccough, pain, increased rapidity of respiration, cyanosis. As was to be expected, the youngest infants proved to be the most responsive to the stimulus, reacting most intensely. It may be stated, therefore, that although we have clinical proof that hydrochloric acid cannot be considered the cause of pylorospasm, a small amount of acid is capable of inducing spastic phenomena in very young infants.

Just a word as to the duodenal secretion. As was to be expected, gastric hypersecretion was found to lead to duodenal hypersecretion, and the cases of pylorospasm manifesting the one form of secretory overactivity were found to evince the other. This condition has previously been termed "duodenal or pancreatic succorrhea." We may add that, just as there is a type of gastric hypersecretion not associated with spasm of the pylorus, so there is a form of pancreatic hypersecretion unaccompanied by gastric hypersecretion or by increased tonicity of this sphincter.

SINGULTUS

Singultus is a frequent symptom accompanying pyloric obstruction, more commonly met with than reports lead one to expect. Although it is evident that this disorder can possess no importance from a diagnostic point of view, it is nevertheless interesting as evidence of an extra-gastric

disturbance of innervation. In very young infants hiccough may be incited at times by passing the catheter through the pylorus, especially a large catheter. As has been noted above, it also may be produced by instilling a few cubic centimeters of normal hydrochloric acid into the stomach (Ginsberg case), and also occasionally by continued pressure on the gastric area. In this connection, mention may be made of similar reflex phenomena encountered in the course of passing the catheter. For example, in many infants, especially new-born infants, touching the pylorus with the catheter causes gagging, which can be brought about as often as the pylorus is impinged on. Or, again, passing the pylorus may lead to what we have termed in our notes, for the sake of brevity, a "pyloric cough" or "sneeze." Certain infants evince these reflex phenomena more readily than others, and in these the singultus, the cough, the sneeze or the gagging may follow each introduction of the catheter. It was found that these reflex phenomena may occur, however, to a far less extent, in connection with the cardiac sphincter. They are of interest in demonstrating the close reflex relationship between the pylorus and other organs, for example the diaphragm and the pharynx.[14] These reflex arcs may have their centers in the spinal cord or even in the medulla, if we are to interpret the cyanosis noted in connection with the introduction of hydrochloric acid into the stomach of the new-born, as a manifestation of impulses conveyed to the respiratory center.

PYLOROSPASM AND ALLIED SPASMS

The classification of pylorospasm requires a few words of comment. From a clinical point of view it would seem that cases of this disturbance should be separated into two groups. The first comprises the cases usually regarded as typical, but which in our experience do not constitute a great majority. In these there is no apparent exciting cause for the disorder; it occurs under ideal feeding conditions; so that for the present they may be designated as primary. The history is familiar to all. In brief, it generally runs as follows: A normal breast-fed infant begins to vomit when a few weeks old; this vomiting increases in intensity and frequency, is markedly projectile in character, and leads to inanition. Generally the outcome is recovery. These cases vary widely in severity; in the moderately severe case the catheter is passed through the pylorus with difficulty, perhaps after a second or third attempt, and

14. This nervous interrelationship between the pharynx and the pylorus seems to be reversible: by exciting spasm of the pharynx (gagging) we induce closure of the pylorus; by passing the catheter through the pylorus we frequently excite gagging.

only with the aid of introducing water into the stomach (accessory method).[15]

The second category of cases is, as we have stated, sufficiently distinct to be classed separately. In these the hyperkinesis and spastic symptoms are secondary manifestations appearing as a complication of various disturbances, the most common of which are disorders of the alimentary tract. The history in this type is generally commonplace, merely reciting that the infant was bottle-fed and thrived until the sudden onset of repeated and forcible vomiting. However, this pylorospasm may supervene in the course of other disorders, as will be seen from the case we shall cite. This case is one of congenital obliteration of the bile ducts, and is quoted as an example of this secondary form of pylorospasm, because it had been repeatedly tested by means of the catheter before spasm developed and was controlled by post-mortem examination (autopsy protocol):[16]

Mary E., 3½ months old, 9 pounds 2 ounces. A patient with congenital obliteration of the bile ducts which had been in the hospital during the latter part of December, 1911. At that time the tube was passed on eight occasions and the cardiac and pyloric sphincters found normal. Free HCl was frequently present.

Feb. 7, 1912. Doing poorly; has been vomiting; vomiting expulsive in nature. Jaundice intense, stools clay-colored. Catheter passed: Congo +++ fluid in stomach. Marked cardiospasm; great difficulty in entering stomach. (Probably food has not entered stomach.) Difficulty in entering duodenum; "accessory method" had to be resorted to. Duodenal feeding with whey.

February 8. Still vomiting, as yesterday. Catheter passed, cardiospasm unchanged, pylorospasm less. Duodenal feeding.

February 9. Doing poorly. Vomiting almost continuous. Cardia and pylorus less spastic. Duodenal feeding.

Feb. 10. Exitus. Autopsy; stomach negative.

Epicrisis: Development of cardiospasm and pylorospasm in a case of congenital obliteration of the bile ducts.

---

15. In passing the duodenal catheter we now regularly make use of what has been previously described as the "accessory method." This consists in allowing warm water to flow into the stomach before the catheter is inserted into the duodenum. Water is drawn up into the glass aspirating bulb after the catheter is inserted to about the 22 cm. mark and, as it is seen to flow into the stomach (which may occur by gravity or by the aid of pressure applied by blowing into the bulb), the catheter is slowly inserted beyond the 25 cm. to the 35 or 40 cm. mark. The use of the water in this connection has several advantages: it not only renders the passage of the catheter easier, but it aids in judging whether the pylorus has been traversed; in the first place by stimulating the bile, so that we are more apt to aspirate a bile-colored fluid; second, through the knowledge that if the end of the catheter is in the duodenum, it is generally impossible to aspirate much of the water which has been put into the stomach, whereas if the end is in the stomach we are able to regain almost the entire quantity. Aspiration should be gentle; nothing is gained by applying forcible suction, but it may lead to slight bleeding. We may add that although the technic of duodenal catheterization is not difficult, nevertheless, like other examinations (for example, the inspection of the ear drum), it requires some practice. It should not be attempted for the first time on a case of pylorospasm.

16. Hess, A. F.: Arch. Int. Med., July, 1912.

From a clinical viewpoint this spastic condition may be termed *secondary pylorospasm,* in contradistinction to the classical *primary pylorospasm.* Usually the secondary cases run a shorter course, are milder, more transient, and offer less obstruction to the passage of the catheter. We have encountered primary cases in which it was impossible to traverse the pylorus, but no case of this intensity in the other group. The peristaltic waves and tumors and the character of the vomiting offer no points of difference.[17] The prognosis in the one type of case depends mainly on the degree of inanition, in the other in a great measure on the primary disorder on which the spasm has been engrafted.

There is a large number of cases which we have not attempted to classify, including both breast-fed and bottle-fed infants — mild cases also characterized by repeated spastic vomiting. We have had an opportunity to pass the duodenal catheter on a considerable number of these patients and in this way to control the dubious diagnosis of pylorospasm. In these cases no obstructive spasm was encountered; in fact, it is solely as the result of negative catheterization that we have not included them under pylorospasm. We do not wish to state unreservedly that the pyloric sphincter is not involved in these disturbances; perhaps further tests will inform us. The following is a typical case:

Nov. 30, 1912. Three months' old infant (seen with Dr. Leopold). Weight 10 pounds 14 ounces. Bottle-fed. Under observation three weeks. For past two weeks projectile vomiting of food or even of water. No visible peristalsis.

Catheter passed three hours after feeding. Ph.R. negative; slight cardiospasm found in first test. Congo ++ fluid in stomach. Catheter passed readily into duodenum; bile, pyloric casts.

Epicrisis: Continued projectile vomiting. Pylorus readily permeable.

ALLIED SPASMS

Is spasm of the pylorus encountered in general disorders of the nervous system? This is a question of interest from a diagnostic, and even more so from a pathogenetic, point of view. When we review our cases from this standpoint, we find that unless caution is exercised, they may be interpreted either for or against this hypothesis. Among the new-born we met with instances in which infants were exceptionally nervous (*e. g.,* Rochman twins, and Rotter, a 3-days-old baby), but the catheter could nevertheless be readily passed into the duodenum. On the other hand, one of the cases which we cited above (Weinstein) as an instance of "latent congenital pylorospasm" was certainly an exceptionally nervous baby. When we consider older infants, the study is simpler, although we may not be able to draw conclusions more sharply. In the course of the past two years the catheter has been passed on

17. The only peculiarity about projectile vomiting in infants is its infrequency. It is as a rule merely the type of vomiting common to adults.

infants suffering from meningitis, tetany, microcephalus, hydrocephalus, and other conditions, where the somatic nerves were involved, as shown by rigidity of the body, exaggerated reflexes, or increased mechanical and electrical irritability. Although in some of these cases it was found that there was an increased spasm of the pylorus, yet the frequent exceptions to this rule preclude the deduction that there is an essential association between the spastic condition of the superficial muscles and that of the alimentary tract. That this association occurs is the experience of all clinicians, as shown by the pharyngospasm and spastic vomiting so characteristic of meningitis. Of this relationship it seems unnecessary to cite specific instances. We shall cite, however, two examples of decreased tonicity where *a priori* we should have been led to expect hypertonicity:

1. Dec. 10, 1911. Gussie Yager, 9 months old. Suffering from pneumococcus meningitis. Spasm of upper and lower extremities, increased knee-jerk, marked trismus, making it difficult to separate the jaws, rigidity of neck, inequality of pupils, *tache cèrèbrale*. No vomiting.

In spite of the general rigidity, the catheter was passed at once into the duodenum and bile obtained.

2. Nov. 24, 1913. Charles Stein, marasmus, spasmophilia. Three months old, 7 pounds 15 ounces. Marked rigidity of arms, legs, neck; knee-jerk clonus.

Ph.R. increased. The catheter was passed directly into the duodenum. Later a No. 20 (F.) catheter passed the pylorus. Bile was obtained in both instances.

Let us reverse the viewpoint and, with this question in mind, survey our cases of pylorospasm. Here we encounter the same lack of uniformity; that is to say, we find pylorospasm where the infant showed no other signs of involvement of the nervous system, or, again, cases in which the infant showed a general nervous irritability (Souter, Malzman), and increased muscular rigidity and ankle clonus (Dede). In general, however, it may be stated that nervous disturbances are found more often associated with these various spasms of the sphincters than can be accounted for unless we grant them a place in the symptomatology of this condition.

Although vomiting and retention of food are valuable clinical symptoms of spasm of the pylorus, they are gross criteria and do not reveal a mild hypertonic condition of this sphincter. Catheterization has convinced us that pylorospasm may be present not only in the new-born but in older infants and yet produce no symptoms. The symptoms depend to a great extent on the persistence of the spasm of the pylorus. If the spasm is tonic in character, we shall have marked obstruction to the outflow of food into the duodenum. However, we may have definite increased tonicity of the pylorus and yet, if this increased spasm relaxes to allow the food to leave the stomach — in other words, if it evinces a clonic rather than a tonic character — there will be little or no gastric

disturbance. Our experience with catheterization of many hundreds of cases leads to the conclusion that, as regards the tonicity of the pylorus, there is no sharp line between the normal and the pathological, but rather a natural gradation such as we find in all functional conditions.

Throughout this paper we have mentioned pharyngospasm and cardiospasm as accompanying spasm of the pylorus or as occurring interchangeably with pylorospasm. There are other spasms of the viscera, and especially of the alimentary tract, which are met with from time to time in conjunction with pylorospasm. In one instance we found laryngospasm, as manifested by laryngeal stridor and retraction of the suprasternal notch (Stark case). In this same case there was anospasm, a condition which certainly occurs more often than is noticed. The spasm of the anal sphincter leads to the production of fissures and then to prolapse of the rectum. One of the new-born infants suffering from congenital pylorospasm had this condition. A case of spasm of the pylorus which we recently examined when it was convalescent also had fissures and prolapse, which must be regarded as the result of Nature's forcible efforts to overcome the spastic anal sphincter.

We are unable from personal experience to state whether enterospasm is frequently associated with pylorospasm. It is certain, however, that some of the infants (*e. g.*, Stark case) who suffer from spasm of the pylorus show increased intestinal peristalsis and also signs of abdominal pain. However, the inexplicable cases of colic which are encountered so frequently among breast-fed infants are generally not associated with gastric symptoms. In this connection we may add that we have read of but one case of intussuception accompanied by pylorospasm.

It is questionable whether, at the present time, we should group these various spasms and secretory disturbances under a title such as "vagotonia," which implies a definite etiological conception. In a recent case which we tested with atropin and epinephrin for a vagotonic condition, the reaction was negative; eosinophilia was also absent. However, whether we accept this anatomical classification or substitute a descriptive clinical term, it is evident that there is a condition manifested by spasmophilia of the alimentary tract, and that pylorospasm is a part of this neurosis and rarely met with as an isolated symptom.

CONCLUSIONS

The foregoing study is based on a considerable number of cases in which an obstruction was found to the passage of the duodenal catheter. As the catheter was able, nevertheless, to traverse the pylorus in all these cases, they were regarded as instances of spastic rather than of organic stenosis. All other cases have been excluded from consideration, including instances of pylorospasm in which the catheter could not be passed.

An effort was made to ascertain the diameter of the pylorus *in vivo,* as previous measurements of this sphincter have been based on post-mortem examination and must be regarded as purely anatomical in nature. As a result of many tests with catheters of various diameters, the conclusion was reached that *the functional circumference of the pylorus in infants 2 to 3 months of age is about 18 mm. and the functional diameter about 6 mm., and that in the new-born these measurements are about 14 mm. and 4.75 mm., respectively.*

Tests on new-born infants showed that even in these there is a distinct individuality in the tonicity of the pylorus. A few showed marked spasticity of the pharynx, of the cardia, of the pylorus, or of more than one sphincter. In other words, cases of *congenital pharyngospasm, cardiospasm and pylorospasm* were encountered. These conditions were found at birth, even before any food had been given, or secondary to some slight alimentary disturbance occurring in the course of the first few days of life. The progress of several of these babies was followed for the first months of life, and it was found that in some the spasm remained latent, but that in others spastic symptoms were manifested in later infancy, such as projectile vomiting and obstruction at the pylorus. Not only was pyloric spasticity encountered at birth, but, on the other hand, *pyloric insufficiency,* especially in cases of pronounced icterus neonatorum, where there is hypercholia; in these cases the bile welled past the pylorus into the stomach, and the catheter met with no resistance in its passage into the duodenum.

An interrelationship was found between pharyngospasm and pylorospasm. In some cases, pharyngospasm led to vomiting immediately after feeding, and in this way prevented milk from entering the stomach.

This same close relationship was found also as regards cardiospasm; and in the same way this disturbance at times prevented the entrance of food into the stomach, resulting in what may be termed *"cardiospastic vomiting."* Cardiospasm is far more frequent in infants than is generally recognized. It may reach even such a degree as to make it necessary to introduce a stylet into the catheter in order to pass the cardia. In mild cases, cardiospasm is able to obstruct fluid poured through the catheter, or to prevent the aspiration of fluid from the stomach. Cases were encountered in which the gastric hypersecretion seemed to be the cause of this spasm of the cardia; however, in others, there was no increase of hydrochloric acid, so that it cannot be considered an essential etiological factor.

Visible peristalsis and peristaltic tumor (pseudotumor) not only are not significant of organic obstruction, but even a spastic obstruction is not necessary for their production. Numerous cases were encountered with pronounced hyperkinesis of this description, where the catheter

met with no obstruction at the pylorus. This is especially true of cases of alimentary intoxication. Furthermore, as these signs may be absent where numerous tests convincingly demonstrate pylorospasm, we must conclude that there is no essential interrelation between obstruction at the pylorus and hyperkinesis. It was possible to produce peristalsis artificially in the new-born by instilling 2 c.c. of 0.4 per cent. hydrochloric acid into the stomach, and in older infants to induce distention or marked ballooning of the stomach by this means. The presence of the catheter did not lead to peristalsis, even where this sign could be brought to view by putting a small amount of liquid into the gastric cavity.

A palpable tumor was not encountered in any case in which the catheter could traverse the pylorus; nor did the presence of the catheter within the duodenum render the pylorus palpable.

Gastric hypersecretion is not the cause of these marked cases of pylorospasm. Cases were tested in which these two conditions were found associated, but also in which spasm existed without the accompaniment of gastric hypersecretion. Although the introduction of a small amount of hydrochloric acid into the stomach of the new-born was not sufficient to produce spasm of the pylorus, it led to marked reflex symptoms, e. g., gagging (pharyngospasm), hiccough, pain, increased rapidity of respiration.

In some cases of pylorospasm, those accompanied by gastric hypersecretion, duodenal hypersecretion or succorrhea was also encountered.

Singultus is a frequent symptom of pyloric obstruction. It may be incited in new-born infants not only by allowing a small quantity of hydrochloric acid to enter the stomach, but by the passage of the catheter through the pylorus; the latter may also lead to pyloric cough. Singultus is interesting in this connection as an evidence of extragastric nervous involvement in this condition.

Pylorospasm should be subdivided into two groups, the *primary* cases, that is to say, those which occur in breast-fed infants without apparent exciting cause (the classical instances), and *secondary pylorospasm*, occurring as a complication of some other disturbance, generally alimentary in nature. The former are the more severe. However, the peristaltic waves and tumors may be equally marked in either.

Mild pylorospasm, cardiospasm and pharyngospasm were at times encountered in tetany and other nervous disturbances, indicating a spasmophilia of the alimentary tract.

Moderate spasm of the pylorus may exist, as proved by catheterization, and nevertheless evince no symptoms (latent pylorospasm), as the symptoms depend in a large measure on the frequency of relaxation of the spasm; that is to say, on its degree of tonicity or clonicity. There

is no sharp dividing line between the normal and the spastic pylorus: tonicity, hypertonicity, spasticity, are merely positive, comparative and superlative terms.

In addition to pharyngospasm and cardiospasm, other visceral spasms were found associated with pylorospasm, e. g., laryngospasm and anospasm, the latter leading to anal fissures and prolapse. Pylorospasm was found rarely as an isolated spastic symptom.

16 West Eighty-Sixth Street.

STUDIES IN CARDIAC STIMULANTS*

I. STRYCHNIN AND CAFFEIN GROUP

WILLIAM PALMER LUCAS, M.D.
SAN FRANCISCO

In considering the therapeutics of cardiac conditions in children, one is rather surprised to find that therapeutic measures have either been handed down or accepted simply from pharmacological experimentation, which is not always an accurate estimate of the clinical value of any therapeutic procedure or of any drug. It is interesting to note the paucity of material which deals with accurate observations of the different drugs used in cardiac conditions. Practically all of the English text-books speak of digitalis and strychnin as the two cardiac stimulants of choice. Digitalis has been tested out both experimentally and clinically, so that there can be very little doubt as to its value.

In regard to strychnin, there is conflicting evidence from the side of the pharmacologist and that of the clinician.

It is still further interesting to note that in none of the German text-books is strychnin mentioned, and it is very seldom used except in America and England. In this country it has been accepted, and it has probably the widest use of any cardiac stimulant.

From a pharmacological standpoint, the effect of strychnin is on the sensory centers, not motor. "The action of strychnin," as stated by Dr. Sollmann, "consists in facilitating the passages of the nervous impulses through the sensory paths so that very slight stimulation leads to exaggerated motor responses." Strychnin in small doses increases the reflex excitability of the spinal cord, so increasing the muscle tone, which in itself brings about certain secondary results, such as (1), increased metabolism; more oxygen is consumed and the CO_2 output is increased, as is also the glycogen consumption increased; (2), there is a tendency to a rise in blood-pressure and an increase in the pulse-rate; (3), temperature is increased. Strychnin causes a stimulation followed by depression of the medullary centers, particularly the respiratory and vasomotor centers. Its effect on circulation in small therapeutic doses is a direct action on the vasomotor and vagus centers, and an indirect action by convulsions, respiratory movements and asphyxia. The action of strychnin on the heart is at first stimulation; but this is only seen

* Submitted for publication Nov. 11, 1913.
* This paper was written from the wards of the Boston Dispensary Children's Hospital.
* Read at the meeting of the American Pediatric Society, Washington, D. C., June, 1913.

when the heart is directly perfused with a strong solution, and it cannot be considered, therefore, as a cardiac stimulant in any therapeutic manner. Indirectly, strychnin may improve the circulation by improving the respiration, or by altering the distribution of the blood without affecting the general blood-pressure. In large doses, it has a marked effect on the circulation. There is a marked rise in blood-pressure due to central vasomotor stimulation aided by convulsions and asphyxia. The respiratory center is stimulated by strychnin, both directly and reflexly, through increased muscular work. Its effect on the respiratory center is important. It removes the depression of the respiratory center. "It should not be used in heart diseases, for if it has any action it increases the work of the heart."[1] The action of strychnin on the respiratory center evidently is much more certain than its vasomotor effect, where the respiratory failure is due to depression or fatigue. Further, Dr. Sollmann says: "Its value is as a temporary bridge; it does not permanently improve the condition of the nervous system; it simply increases its irritability." Such is the opinion of one of the foremost pharmacologists of this country.

From clinical studies we have diversified findings. Hirschfelder[2] made some tests on normal persons with normal hearts, and found that with doses as high as ¼ of a grain hypodermically there was no effect on maximal or minimal pressure, pulse-rate or respiration; and from single doses, scarcely any increase in reflexes. Hirschfelder quotes Dr. P. D. Cameron on some experimental tests that he made on the effect of strychnin on cardiac tonicity. Cameron found 1/4,500 grain per pound, or .00003 grams per kilo, which corresponds to about 1/30 grain hypodermically for a man, always produces an increase in the tonicity of the heart muscle, though without affecting the force of the beat or markedly changing the maximal pressure. The mean and minimal pressure are usually slightly increased by 10 to 15 mm. mercury, and usually the pulse-rate was a little slow. Larger doses increase the systolic output, raise the blood-pressure, slow the heart and increase the tonicity. Hirschfelder[2] further says (p. 181) that strychnin stimulates both the vasomotor and vagus centers, hence raises the blood-pressure and slows the pulse-rate.

In 1903, Henry W. Cook,[3] and Henry Cook and John B. Briggs[4] give their observations on the clinical value of Blood-Pressure. They say:

---

1. Sollmann: Text-book of Pharmacology, p. 159.
2. Hirschfelder: Diseases of the Heart and Aorta, p. 182.
3. Cook, Henry W.: Clinical Value of Blood-Pressure Determinations as a Guide to Stimulation in Sick Children; Am. Jour. Med. Sc., cxxv, p. 433, and Johns Hopkins Bull., Feb., 1903, p. 38.
4. Cook, Henry W., and Briggs, John B.: Clinical Observations on Blood-Pressure, p. 512; Johns Hopkins Hosp. Rep., 1903, ix.

Strychnin was found to have a more positive effect on the blood-pressure. Hypodermic doses of 1/400 of a grain for infants, and 1/200 of a grain during the second year, and 1/100 during early childhood as a rule produced a rise in blood-pressure in ten to twenty minutes, which was well maintained for two to six hours as long as the child was not moribund. Children that do not need stimulation do not show these marked responses to strychnin, nor do the moribund when the vasomotor center is insensitive to stimulants. It is the large intermediate class of toxic and marantic conditions accompanied by low tension pulse-pressure met with in pediatrics where carefully regulated stimulation is so satisfactory in its beneficial results.

Cook and Briggs, working with adults, state that strychnin given hypodermically in doses of 1/60 to 1/10 of a grain produces a rise in blood-pressure, the onset of which is somewhat delayed as compared with that sometimes seen following the administration of alcohol by mouth, whose increase is not so abrupt, but which is maintained for a much greater length of time, varying in average cases from one to four hours. This rise in blood-pressure is distinctly seen to be accompanied, in the majority of patients carefully observed, by an improvement in the patient's general condition. Again:

On the whole, strychnin is by far the most satisfactory cardiac stimulant for long routine administration, the maintenance of a satisfactory blood-pressure level free from intervals of depression being most easily accomplished by its use in appreciable doses.

They found a rise in blood-pressure from 80 to 120 mm. Hg after 1/20 grain not extraordinary. They believe in starting with large doses when there is a low pressure, and sustaining the increase which they obtain with smaller doses at regular intervals. They give three groups of cases in which strychnin in moderate and large doses fails to raise the arterial blood-pressure: (1), moribund cases (in the following work there are none of this class); (2), cases not really in need of stimulation — relatively normal individuals; (the question might be raised whether cardiac cases can be classed in this group; if so, perhaps the cases which follow might be said not to have needed stimulation); (3), cases in which the blood-pressure is already being maintained at stimulation level of the individual case. In these three groups, they found that a single dose usually had no effect. Their findings are in marked opposition to the statements which Sollmann made: that strychnin is "only to be used to bridge over some acute respiratory or cardiovascular failure; not for continuous use, and that it is not a cardiac stimulant."

Dr. R. C. Cabot[5] used strychnin in thirty-one cases of typhoid fever, four cases of pneumonia and fifteen cases with a variety of diagnoses. He gave a total daily dose of about 1/8 grain, but sometimes 1/6. He took 5,000 blood-pressure measurements, and comes to the conclusion that the total result was negative. He says, "I have been unable to

5. Cabot, R. C.: Proc. Assn. Am. Physicians, 1904, p. 22.

convince myself that strychnin exerts any influence on the blood-pressure in febrile cases when given in the doses as mentioned."

James McKenzie[6] states, "Strychnin acts probably on the nervous system, and by producing some exhilaration proves useful in cases in which a temporary exhaustion causes distress. But it cannot be too strongly insisted on that, though it is commonly employed in extreme cases of the most diverse kind — for instance, where there is a sluggish ventral contraction in heart-block — its usefulness is very doubtful and of a limited kind, and should not be relied on in cases of real heart exhaustion."

David Marvin,[7] in giving the results of some clinical observations which he had made with strychnin on medical students, says: "I believe that this evidence, obtained from thirty observations with varying doses, is conclusive proof that strychnin when given in the above doses by hypodermic injections, does increase blood-pressure in the normal man," the doses being 1/40, 1/30 and 1/20 gr. He found no appreciable effect on the rate of respiration; he found that the pulse-rate was slowed five beats from 1/40 gr.; that the pulse-rate was slowed seven beats from 1/30 gr., and the pulse-rate was slowed eight beats from 1/20 gr. The maximum effect was reached at the end of forty minutes from 1/30 and 1/40 gr.; the effect of 1/20 gr. lasted several hours. The pulse-pressure showed an increase of 3 mm. Hg from 1/40 gr., 13 mm. Hg from 1/30, and 8 mm. Hg from 1/20 gr. His conclusions are that there is no effect on the respiration; there is a slowing of the pulse from all doses; a marked increase in blood-pressure from a thirtieth and a twentieth of a grain, and practically none from a fortieth.

L. H. Newburgh,[8] in some work carried out this past year (1913) at the Massachusetts General Hospital on patients suffering from cardio-vascular conditions, found that strychnin given in the ordinary doses has no effect on blood-pressure, pulse, respiration, cardiac conditions or in the way of general improvement.

The only observations on children are those by Cook previously referred to. He states that the blood-pressure during the first few months of life is from 70 to 75 mm. Hg; from six to twelve months, 80 to 85; that below 60 is rare during the first year, except in cases in which there is marked need for stimulation. During the second year, the blood-pressure ranges from 80 to 90 mm. Hg; during the third year from 90 to 100, and from the third to the tenth year, 85 is moderately low, 75 is low and 65 very low. He found that crying and restlessness raised the blood-pressure 5 to 10 mm. Hg. He found that the highest pressure

6. McKenzie, James: Hare's Modern Treatment, ii, 123.
7. Marvin, David: Arch. Int. Med., 1913, vii, No. 4, p. 422.
8. Newburgh, L. H.: Paper read before the New England Pediatric Society, March 29, 1913, as yet unpublished, so no direct quotations are possible.

occurred between 4 and 7 o'clock at night, and the lowest probably between 3 and 7 a. m. My experience agrees with these findings. In his cases, all of whom were infants, there were no cardiac cases. Judging from his charts, strychnin was usually used in conjunction with, or followed very closely by, digitalis; and if the two were separated, it was not enough so as to be able to separate their effects. In his Chart 5 there is, in fact, a fall of blood-pressure after strychnin instead of a rise, the rise occurring after digitalis. In Chart 6 there is a rise of 10 mm. Hg after strychnin in comparatively small doses. This was not a cardiac case.

I have been able to watch the effects of strychnin in nine cases during the past few months. All these cases were the result of endocarditis occurring in the course of rheumatic fever infection. The methods of carrying out the observations were mainly as follows (the detailed account will be found in the protocol of each case):

After admission to the hospital the children were given very little or no medication the first few days, sometimes for longer periods, to see what effect the rest would have on their general and cardiac condition. They were then given some one of the cardiac stimulants for a definite period. Some of the cases were put on digitalis, and kept on this drug for several weeks before a trial of strychnin was made; and while the strychnin was being given, the patients received no other cardiac stimulant. Strychnin was given for a period of from two to four days, in varying doses, and its effect watched closely and compared with their average picture as relating to cardiac condition, pulse, respiration, blood-pressure, also their general improvement, appetite, freedom from pain, improvement in restlessness, etc., when simply resting in bed or resting in bed plus digitalis. In this way, it was hoped that some conclusions as to the effect of strychnin in such cases could be fairly accurately made. I very quickly found that strychnin is practically of no avail when given in the ordinary doses which are prescribed for children. (See J. P.'s chart.) Even in adult doses of 1/50, 1/40 or 1/30 gr., there were no appreciable effects. It was only when doses amounting to 1/10 grain or more were given (either at one dose or in quick succession of smaller doses) that any appreciable effect was noted; and then the effect appeared within thirty minutes and disappeared within two hours.

In four cases in which I have studied caffein sodium benzoate as a stimulant, and which I am carrying on further at present, in the ordinary dose of ¼ or ½ grain hypodermically or by mouth, I could observe no effect; only in doses of two or more grains hypodermically was there any appreciable effect, and then the effect was very small and of short duration. (See Charts 4, 12, 15, 16, 17.)

From these observations I conclude that strychnin and caffein in the doses usually used for cardiac stimulation are of no value. It is only

when the larger, maximum doses are used that we get any effect, and the effect is usually then of short duration. Whether this is a beneficial effect is even questionable in children with cardiac involvement. It may well be, as Dr. Sollmann suggests, that strychnin stimulation makes the heart work harder, and that it should not be used as a cardiac stimulant. Its effect on respiration in the conventional dosage is of no value; in larger doses, it does appear to slow the respiration up to the point where there are toxic symptoms (slight twitchings) of increased hypersensitiveness of the reflex arc, or when the heart action becomes more rapid and the blood-pressure falls.

CASE REPORTS

CASE 1.—Margaret M., aged 15 years, hospital No. 1198, admitted April 27.

History.—Family history is negative. Patient had had measles and whooping-cough. She was subject to frequent sore throat. She had the first attack of rheumatic fever one year previously. She was treated in the out-patient department for several months and improved until she started to school in September. She grew gradually worse until in October when she was sent to a convalescent home. There she remained until she was admitted to the hospital, April 27. She had been doing well until a week before admission when she contracted severe tonsillitis with high temperature. The temperature ran as high as 103 F. Her cardiac attack became markedly worse; there were marked dyspnea and precordial pain.

Physical Examination.—Physical examination showed a well developed but poorly nourished girl; very pale, with marked dyspnea and slightly cyanotic. She had an anxious look. Her throat was injected and her tonsils were large; there was no exudate. There was a marked precordial bulging and precordial pulsation was visible throughout the anterior thorax. Apex beat was in the sixth interspace 2 inches outside the nipple line. The left border was in the mid-axillary line. The right border was 4 cm. from the mid-sternal line. Heart action was very irregular. The sounds were weak and both systolic and diastolic murmurs were present. It was impossible to get an accurate blood-pressure reading.

Patient was put to bed and given ⅛ gr. morphin, and ice bags were applied to her heart. She was nauseated and vomited easily. Her pulse on admission was 120; respirations 30. By May 2 the patient had quieted down considerably, the pulse remained between 80 and 90, and respirations between 20 and 30.

Cardiac Condition.—If anything her heart was more irregular and her pulse was fuller. Cardiac area was the same size as on admission. She received ⅛ gr. morphin as needed for pain two or three times in twenty-four hours.

On May 2 strychnin was given, 1/30 gr. by mouth every three hours. Her blood-pressure on May 2, before starting strychnin, was between 95 and 100 mm. Hg. It was very hard to read accurately.

May 4 her respiration ranged between 22 and 26, radial pulse between 112 and 120; apex pulse-rate between 130 and 140. Blood-pressure on May 4 at 12 o'clock noon was 100 mm. Hg; at 11 p. m. it was 94 mm. Hg. On May 5 there was no change in the respiration or radial pulse. Blood-pressure at 9 a. m. was between 95 and 100 mm. Hg; at 2 p. m. between 105 and 108; at 7 p. m. between 106 and 110.

May 6 she said she felt better. The heart action was no better, still very irregular, and the heart sounds were indistinct. The size of her heart was the same as on admission. The ice bags were continued. Morphin was given only once in the previous twenty-four hours. Respiration ranged between 24 and 30, radial pulse between 96 and 116, and apex beat between 140 and 150. At 9 a. m.

the systolic blood-pressure was 105; at 3 p. m. systolic blood-pressure was 102 mm. Hg. At 5 p. m. it was 104.

On May 7 there was no change in her cardiac condition. Blood-pressure ranged throughout the day between 103 and 107 mm. Hg. There was no change in the respiration or pulse.

May 8 the systolic blood-pressure ranged between 100 and 106 mm. Hg.

May 9, it ranged between 100 and 104 mm. Hg.

May 10, it ranged between 100 and 104 mm. Hg.

There was no improvement during this period of eight days of strychnin treatment. She had received throughout this period 1/30 gr. every three hours. She

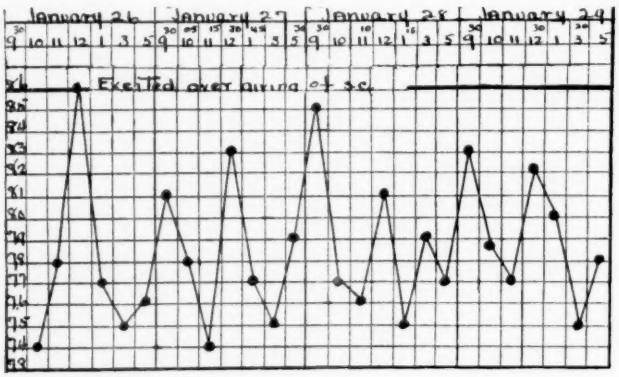

Chart 1, Case 2.—Curve showing systolic pressure in the case of M. M. under strychnin, hypodermically, at 9 a. m., 12 noon, 4 and 8 p. m. for a period of four days. Aspirin, 5 gr., every three hours (six doses in twenty-four hours) and hot baths at 7:30 a. m. and 6 p. m.

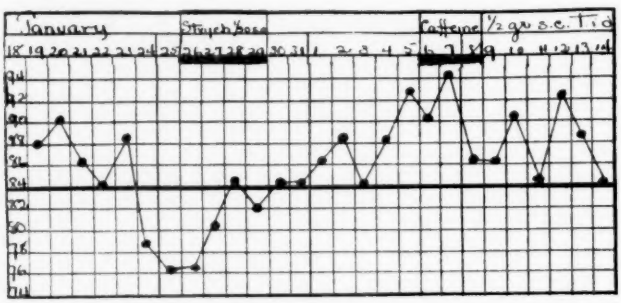

Chart 2, Case 2.—Curve showing systolic pressure in the case of M. M. under strychnin and caffein.

was still markedly dyspneic and at times cyanotic and nauseated. The heart sounds were no clearer and the irregularity was as marked as at entrance. She was having such pains that the ice bag was kept on constantly but she had not received any morphin since May 6.

On May 10 digitalis was started, 5 mm. every four hours, continuing with the strychnin until May 18, when the strychnin was omitted and the digitalis, which had been gradually increased to 10 min. four times a day, was changed to digipuratum, one tablet every six hours for two days, then one tablet three times a day for two days, and then one tablet twice a day. She improved somewhat on digitalis so that she was not nauseated. She had a better appetite and felt

generally relieved. Her cardiac condition, except for the slowing of the rate, remained about the same as on admission. The heart was no smaller and the cardiac action was still markedly irregular. The temperature varied between 100 and 97 F.; the pulse between 60 and 80, and the respiration between 20 and 30.

At this point she was taken home, against advice, to be treated by the district physician.

CASE 2.—M. M., 8 years old, hospital No. 1139; admitted January 18.

History.—Never very strong; had pneumonia, followed by measles a year before present trouble started. She had not been well since. Present illness started three weeks before admission to the hospital, with noticeable choreic movements.

Physical Examination.—A pale, emaciated child, with marked involuntary movements. Teeth were in poor condition; both tonsils were large, showing marked chronic infection. General glandular system was slightly enlarged.

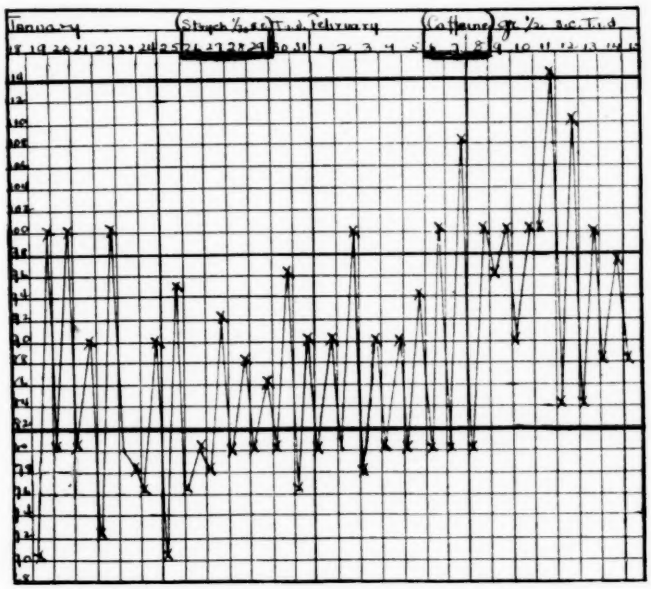

Chart 3, Case 2.—Pulse curve in the case of M. M. under strychnin and caffein.

Heart.—Point of maximum impulse in the fourth interspace, 8 cm. to the left of the midsternal line; left border 9 cm. to left of midsternal line in the fourth interspace, and 10 cm. in the fifth interspace. Right border 2.5 cm. to the right of the midsternal line in the second interspace. First sound at the apex was accompanied by a soft blowing murmur, which could be heard well into the axilla and back. Radial pulses were symmetrical and synchronous, small volume, low tension. With Nicholson's sphygmomanometer the systolic pressure was 80 to 90 mm. Hg. Diastolic pressure varied between 55 and 65, and continued between these limits through the patient's stay in the hospital (twenty-eight days), with the exception as noted in the charts.

Treatment.—The treatment consisted of 40 gr. aspirin a day for the first two days. She was given hot baths, starting at a temperature of 100 F. and gradually increasing the temperature of the water until it reached 110 to 115 F. The length of the baths ranged from half an hour to three-quarters of an hour, gentle friction being given throughout the bath.

January 26 she was put on strychnin. Her temperature from her admission until strychnin was started on January 26 ran between 98 and 99 F.; her pulse between 80 and 100, and her respirations between 20 and 30.

She received 5 gr. of aspirin every six hours — six doses in thirty-six hours. For the chorea she received hot baths at a temperature of 100 to 110 F. twice a day, with gentle friction. She was given 1/30 gr. strychnin on January 26 at 9 a. m. The systolic pressure at that time had ranged between 80 and 100 mm. Hg. Her systolic pressure at 10 a. m. was 74 mm. Hg.; at 11 a. m. it was 78, and at 12 m. it was 86. At this time she was somewhat excited over the prospect of her second injection. At 1 o'clock p. m. the pressure had fallen to 77 mm. Hg; at 3 o'clock to 75 and at 5 o'clock to 76, one hour after her third injection of 1/30 gr. of strychnin.

On January 27 at 9:30 a. m. her pressure was 81 mm. Hg.; at 10:05 a. m. it was 78, and at 11:15 a. m. it was 74; at 12:30 a. m. it was 83, and at 1:45 p. m. it was 77. At 3 p. m. it was 75; at 5:30 it was 79.

She received 1/30 gr. at 9 a. m., 12 m., 4 and 8 p. m.

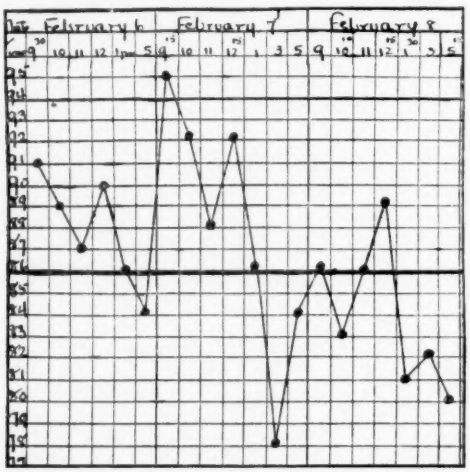

Chart 4, Case 2.—M. M., aged 8 years. Hospital No. 1139. Curves showing systolic pressure under caffein sodium benzoate, ½ gr. hypodermically at 8 a. m., 12 noon and 6 p. m. during a period of three days.

January 28 at 9:30 her systolic pressure was 85 mm. Hg. At 10 a. m. it was 77; at 11:10 a. m. it was 76, and at 12, noon, it was 81. At 1:15 p. m. it was 75. At 3 it was 79, and at 5 it was 77

She received on this day also hypodermics at 9 a. m., 12, noon, 4 and 8 p. m.

On January 29, the last day of this series, she received 1/30 gr. of strychnin at the same hours as on the previous day.

At 9:30 a. m. her systolic pressure was 83 mm. Hg; at 10 it was 78, plus; at 11 a. m. it was 77; at 12:30 p. m. it was 82; at 1 p. m. it was 80; at 3:20 p. m. it was 75, and at 5 o'clock it was 78.

Through this period of four days, from January 26 to 29, her pulse remained constantly between 75 and 85 and her respirations between 20 and 30. She complained of no headache and there was no increase in the twitching. The urine throughout this period was normal. She then received a rest of seven days through which she received only aspirin and hot baths. She was then put on caffein sodium benzoate, ½ gr. subcutaneously, three times a day — 8 a. m., 12 noon and 6 p. m.

From January 26 to February 6 the general average of systolic pressure had risen from 84 mm. Hg to approximately 90 mm. Hg.

On February 6 at 9:30 a. m. she received her first hypodermic injection of caffein; the systolic pressure was then 91 mm. Hg. At 10 a. m. it was 89, at 11 it was 87, at 12 noon it was 90, when she received her second dose. At 1 p. m. it was 86; at 5 p. m. it was 84. The next morning at 9:15 it was 95, which was one and one-half hours after her fourth injection. At 10 it was 92; at 11 it was 88; at 12:15 p. m. it was 92; at 1 it was 86; at 3 it was 78; at 5 it was 84. On February 8 she also received three injections — 8 a. m., 12 noon, and 6 p. m.

At 9 a. m., February 8, the systolic pressure was 86 mm. Hg.; at 10:10 it was 83; at 11 it was 86; at 12:15 p. m. it was 89 (fifteen minutes after her second injection for that day). At 1:30 it was 81 mm. Hg; at 3 it was 82, and at

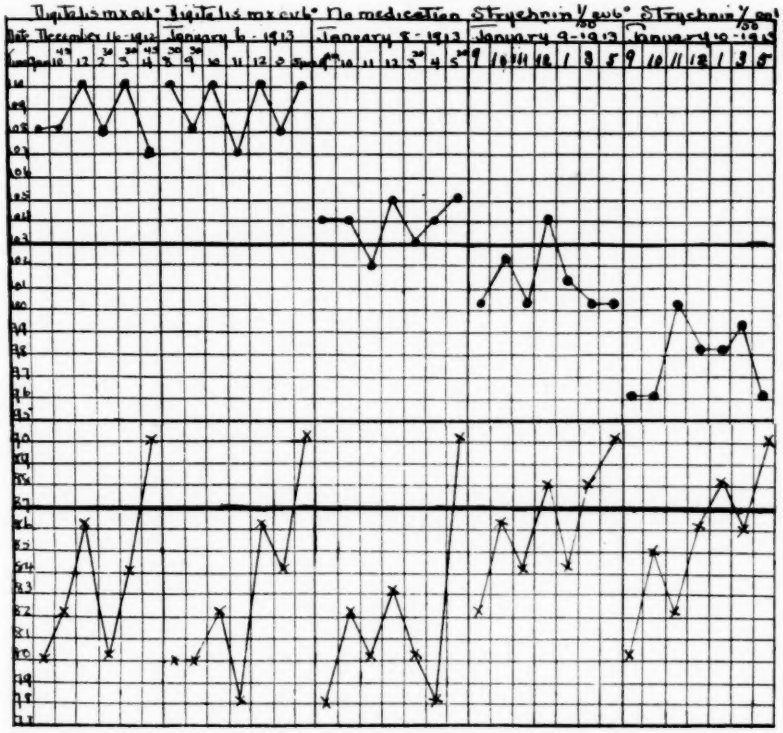
Digitalis max. Digitalis max. sub. No medication Strychnin 1/60 Strychnin 1/30
January 6 - 1913
January 8 - 1913
January 9 - 1913
January 10 - 1913

Chart 5.—M. B., aged 13 years. Hospital No. 1032. Upper curves show systolic pressure and lower curve, the pulse-rate under digitalis, under no medication, and under strychnin.

5:15 it was 80. On the whole there was a slight decrease while on caffein. There was no increase in her choreic condition. In fact that improved steadily under hot baths. There was no increase in the pulse, which continued between 80 and 100, and her respirations continued between 20 and 30. The temperature throughout both periods of strychnin and caffein remained between 98 and 99 F.

CASE 3.—J. P., aged 10 years, hospital No. 890.

History.—The patient was always well until one and one-half years ago, when she developed rheumatic fever, with both arthritis and endocarditis. She was admitted to the hospital April 20, 1912.

Physical Examination.—The physical examination showed a well developed but poorly nourished girl; comfortable only in sitting posture; marked precordial bulging, and diffuse cardiac impulse. Right border 3 cm. from midsternal line in third interspace, and left border in midaxillary line. Palpable thrill; presystolic, diastolic and systolic murmurs; pulse irregular and small.

Treatment.—She was put to bed, given aspirin, 40 gr. a day, and tincture digitalis from 9 to 30 min. a day till June 10, when digitalis was discontinued. The heart had diminished in size, the left border being in the anterior axillary line; pulse had been regular and was the typical digitalis-stimulated pulse. General condition was good. She was comfortable and appetite was good.

She was given strychnin, 1/100 gr. every six hours by mouth, from June 11 to 17. By June 14 the pulse had become irregular and the heart had dilated out to the midaxillary line, but she still felt comfortable and her general con-

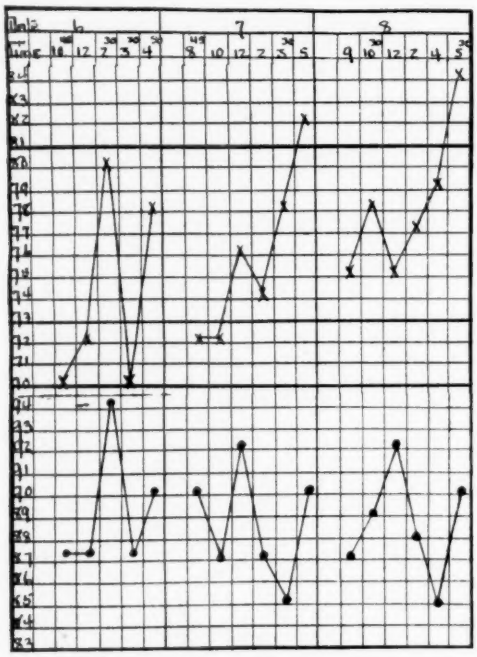

Chart 6.—M. F., aged 12 years. Hospital No. 1071. Upper curve shows pulse-rate and lower curve systolic pressure under strychnin, 1/50 gr., hypodermically, three times a day for a period of three days.

dition remained good. By June 17, however, she was uncomfortable, pulse more rapid and she was nauseated; heart was out to the midaxillary line. She was put back on digitalis and by June 20 was feeling much better.

Effect of Strychnin: The pulse grew irregular and more rapid; blood-pressure fell; respiration at first fell, then rose; general condition not so good, nauseated, loss of appetite, precordial pain.

CASE 4.—M. F., aged 12, hospital No. 1071.

History.—The patient had always been a sickly child. Had measles at 3 years; jaundice at 4 years; erysipelas at 8 years. Had sore throat frequently. Was admitted to the hospital Nov. 5, 1912, with an indefinite history of pain in chest for "a long time."

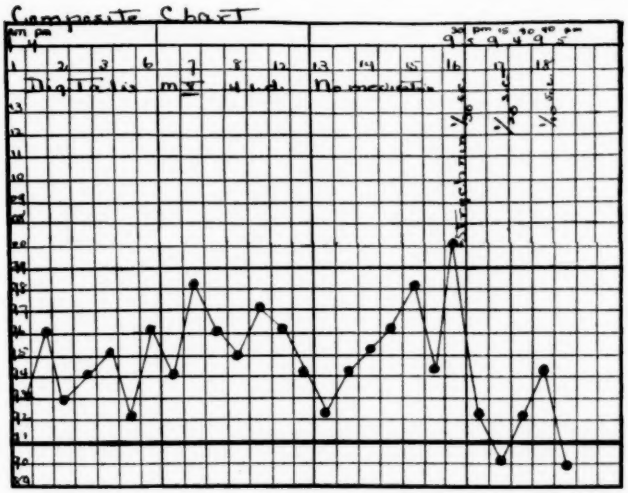

Chart 7.—J. McC., aged 13 years. Hospital No. 1138. Composite chart showing blood-pressure curve under digitalis, no medication, and under strychnin.

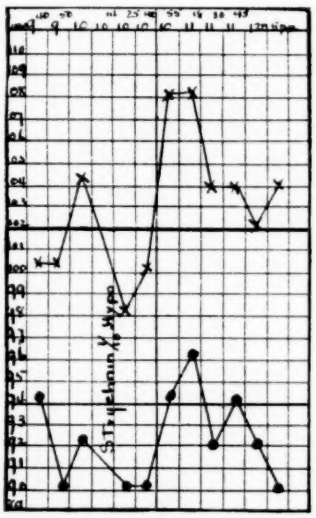

Chart 8.—J. McC. Upper curve shows pulse-rate; lower curve systolic pressure under strychnin, 1/10 gr., hypodermically.

Physical Examination.—The physical examination showed a fairly well developed and nourished boy, very pale and sallow. Moderate general glandular enlargement. Throat showed signs of repeated inflammation. Teeth were very poor.

Heart.—Maximum impulse seen and felt outside nipple line in fourth interspace. Left border 11.5 cm. from midsternal line in the fifth interspace, and right border 5.5 cm. from the midsternal line in the third interspace. The normal first sound was obliterated by a loud blowing murmur heard all over the precordia and transmitted to the axilla and back. There was also a murmur heard during diastole to the left of the sternum in the third and fourth interspaces. On admission his systolic blood-pressure was 90 mm. Hg, according to

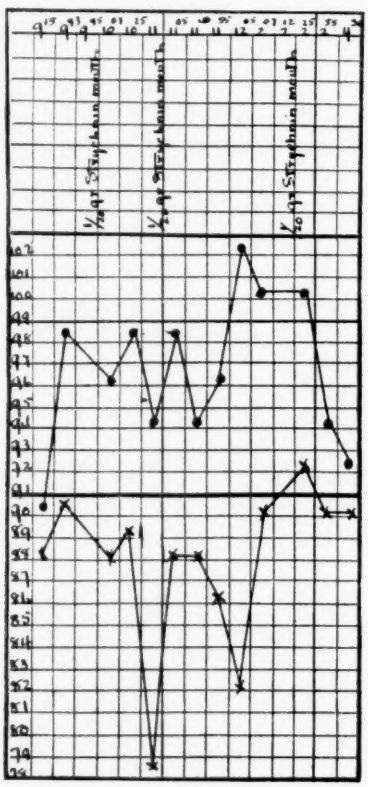

Chart 9.—J. McC. Upper curve shows systolic pressure, lower curve the pulse-rate under strychnin.

Nicholson's sphygmomanometer; pulse-rate 88; pulse was very soft and compressible, and not of very good quality. His peripheral circulation was poor; hands and feet were cold, purplish, subnormal. He remained in the hospital from Nov. 6, 1912, to January 16, 1913.

Treatment.—For the first two months he was given absolute rest in bed. For the first week 25 gr. of aspirin was given in twenty-four hours; for the first three weeks digitalis 20 to 32 min. in twenty-four hours. His systolic pressure during this period remained between 85 and 95 mm. Hg. After an interval of three days without medication, on December 6 he was put on strychnin, 1/50 gr. three times a day, hypodermically, and later four times a day, which gave him from

1/12 to 1/15 of a grain in twenty-four hours. He was kept on this for three weeks. During this time his general condition improved, as would be expected from the continued rest in bed. There was no change in his pulse-rate nor in his pulse-pressure that could not be attributed to the rest in bed. His peripheral circulation improved with the improvement in his heart. The heart area had lessened by 1 cm. on the left, and 5 cm. on the right. During the next three weeks he was given no medication except a stomach tonic which contained

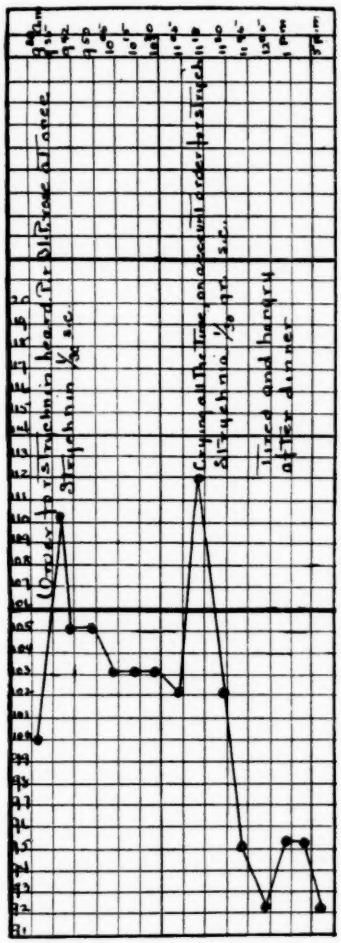

Chart 10.—J. McC. Blood-pressure curve showing the results of emotional excitement.

5 minims tincture nux vomica. During this period he was allowed up in a wheel chair for half-hour periods twice daily. His general condition improved more rapidly than at any other period. His heart diminished in area 1.5 cm. on the left and 1 cm. on the right. Pulse and peripheral circulation improved as rapidly as at any time and he was discharged to continue treatment under careful supervision. Before going out he was put on digitalis, 5 minims, four times a day.

CASE 5.—M. B., hospital No. 1032. Aged 13 years.

History.—This child was an old heart case and had been in the hospital several times with broken compensation. She was on digitalis continuously. She had been in the hospital from Sept. 27, 1912, to the time of the present study, which was Jan. 9 and 10, 1913. She had improved so that her heart was compensating enough to allow her to be in a wheel chair for half hour twice daily. Throughout this period she had been receiving 10 min. of the tincture of digitalis every six hours. Her pulse had remained, for a month previous to the giving of the strychnin, between 80 and 100; her respiration between 20 and 30; her temperature had been slightly subnormal, running between 97 and 98.4 F. Her general condition was good.

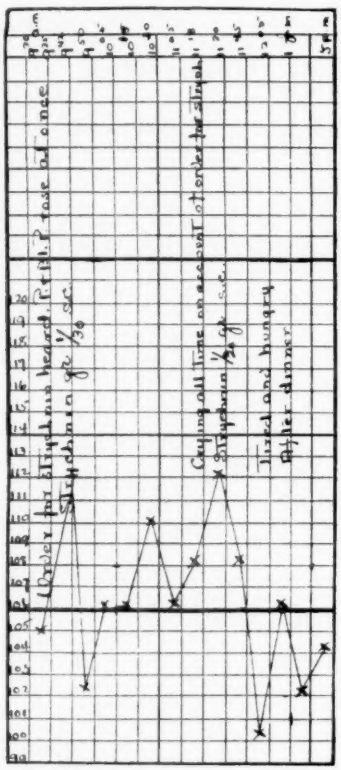

Chart 11. J. McC. Pulse curve under emotional excitement while the patient was taking strychnin.

On the evening of January 7 all medication was stopped. Through January 8 she received no medication at all and was allowed up for her two half-hour periods. On January 9 she received 1/30 gr. of strychnin four times in the twenty-four hours starting at 9 a. m. Her systolic pressure had been running between 108 and 110 mm. Hg, while she was on digitalis. During the period of twenty-four hours when she received no medication her pressure had fallen to between 102 and 105 mm. Hg. At 9 a. m. on January 9, when she received her first hypodermic of 1/50 gr. strychnin, her systolic pressure was 100 mm. Hg. At 10 it was 102; at 11 it was 100; at 12 noon it was 104; at 1 p. m. it was 101; at 3 p. m. it was 100, and at 5 p. m. it was 100 mm. Hg.

January 10 she continued receiving every six hours 1/50 gr. strychnin, 4 doses in twenty-four hours. At 9 a. m. her systolic pressure was 96 mm. Hg; at 10 it continued at 96; at 11 it was 100; at 12 noon it was 98; at 1 p. m. it was 98; at 3 it was 99, and at 5 it was 96.

Her pulse, which while she was on digitalis had been constantly between 80 and 100, had dropped to between 75 and 80. On the whole during the two days in which she received the strychnin it remained between 80 and 90. There was no change in her temperature. She began to complain of precordial pains, from which she had been free for over a month. She was taken off of strychnin and put on digitalis, her pulse and blood-pressure returning to her normal again within forty-eight hours.

CASE 6.—J. McC., aged 15 years, hospital No. 1138; was admitted January, 1913.

History.—The patient was never a very robust boy. He had had rheumatic fever since 1909, and had had endocarditis since 1910. Had a double mitral lesion. Heart had not compensated enough for him to be able to do very much, i. e., go to school, for over a year. He was admitted to the hospital with failure of compensation, cyanotic, peripheral circulation very poor, pulse small, regular, but of very poor quality.

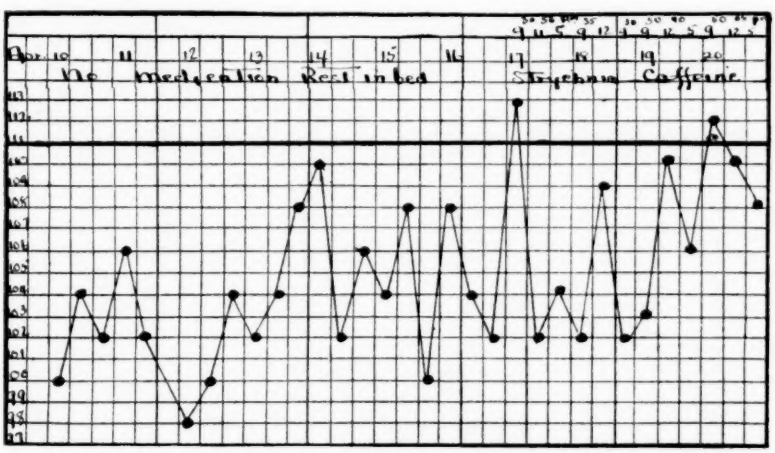

Chart 12.—L. P., aged 2 years. Hospital No. 1169. Systolic pressure curve under rest in bed, without medication, under strychnin and under caffein.

Heart.—Left border 12 cm. in fifth interspace, and right border 8 cm. in third interspace. On admission there was a definite presystolic thrill. The heart sounds were obscured by a presystolic and systolic murmur heard all over the precordia; second pulmonic greatly accentuated.

Treatment.—The patient was put to bed for several weeks, without any marked improvement in his general or cardiac condition, and then he was put on tincture digitalis for two months. During this period he remained in bed, except for two hot tubs daily for the peripheral circulation. He was given several courses of aspirin, 40 gr. a day, for four to seven-day periods. At this time his general condition had improved. He could sit up in a wheel chair with comfort. The peripheral circulation was very much better, and the heart area had diminished by 1 cm. on the left, and 5 cm. on the right. The pulse rate varied, ranging between 85 and 100. Systolic blood-pressure ranged between 95 and 105 mm. Hg. Then there was a period of three days without any medication. As far as could

be seen, during these three days he remained in exactly the same condition as he had been, there being no change in general condition, peripheral circulation, heart measurements, quality of heart sounds, pulse rate, or systolic blood-pressure.

His first period of strychnin treatment was begun on March 17 when he received 1/20 gr. at 9:45 a. m. At 9:15 systolic pressure was 90 mm. Hg; at 9:43 it jumped to 98, presumably from anticipation of the subcutaneous injection, as he was nervous over the preparation of his hypodermic. At 9:45 1/20 gr. strychnin was given. By 10.07, twenty minutes after the hypodermic had been

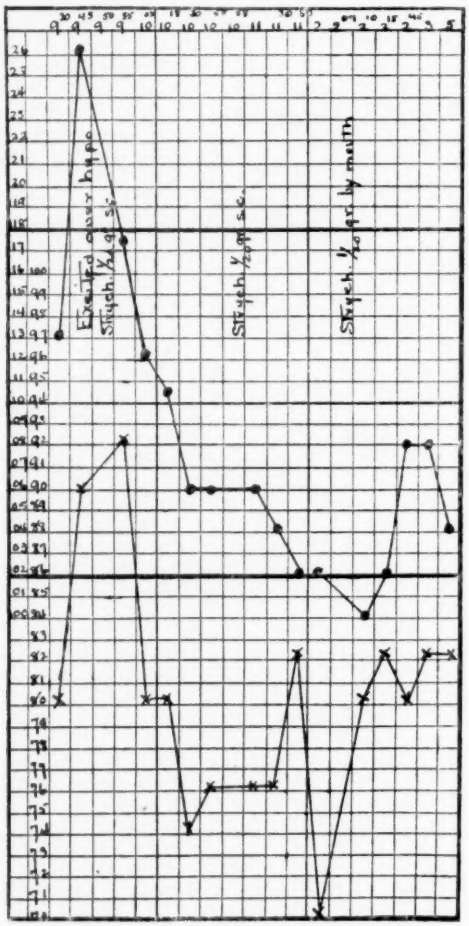

Chart 13.—L. P. Upper curve shows systolic pressure, lower curve the pulse-rate under strychnin. Both curves show a sharp rise during the excitement occasioned by the anticipation of a hypodermic injection of strychnin.

given, the pressure was 96; at 10:25 it was 98. At 11 he received another 1/20 gr. of strychnin subcutaneously, his systolic pressure being 94 mm. Hg. At 11:05 systolic pressure was 98; at 11:40 it was 94; at 11:55 it was 96; at 12:05 noon it was 102; at 2:07 p. m. it was 100. At 2:12 he received another 1/20 gr. subcutaneously. At 2:25 it was 100 mm. Hg; at 2:55 it was 94, and at 4:30 it was 93. He was then left with no medication for almost a month, when on April 16th he was started on strychnin again.

During this interval of a month his pulse ranged between 85 and 100; blood-pressure between 95 and 105 mm. Hg. He was then allowed up and around in a wheel chair. April 16 at 9:30 a. m. his systolic pressure was 106. At 9:35 he heard the order for strychnin. The blood-pressure rose to 110. He was given the strychnin 1/30 gr. at this time and at 9:42 his blood-pressure had fallen to 105 mm. Hg. At 9:50 it was still 105; at 10:05 it was 103; at 10:15 it was 103; at 10:30 it was 103; at 11:05 it was 102. At 11:18 strychnin was again ordered and the systolic pressure rose to 112, probably because he started to cry. Strychnin was given at this time and by 11:30 his pressure had fallen to 102. He had stopped crying. At 11:45 it had dropped to 95; at 12:05 noon it had dropped to 92. At 1 p. m. it was 95 and remained so during the afternoon until 5 o'clock, when it had dropped to 92 mm. Hg. His pulse had changed with the systolic pressure throughout the day, as seen in the chart.

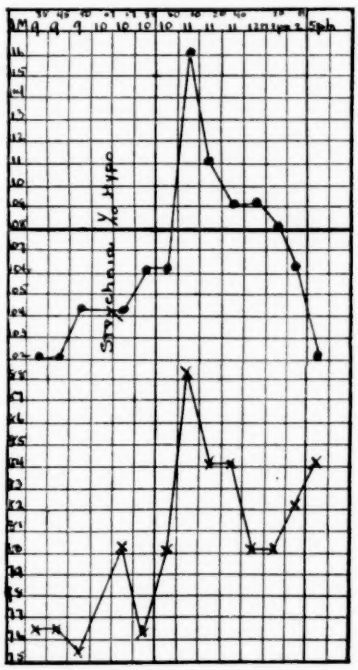

Chart 14.—L. P. Upper curve shows systolic pressure, lower curve shows pulse-rate under strychnin, 1/10 gr., hypodermically.

April 17 the dose of strychnin was increased to 1/20 gr., which he received at 9:45 a. m., 11 and 2:12 p. m. The effect of this is seen on the chart. There was a slight rise between 11 a. m. and 2 p. m. from an average of 98 to 102, remaining there until after his third injection, when it dropped back to 92 mm. Hg. There was very little effect on the pulse.

April 18 at 9:40 a. m. his systolic pressure was 94 mm. Hg; at 9:50 it was 90; at 10 it was 92. At 10:14 he received a 1/10 gr. strychnin hypodermically. At 10:25 a. m. the systolic pressure was 90 mm. Hg; at 10:40 it was 90; at 10:55 it was 94; at 11:18 it was 96; at 11:30 it was 92; at 11:45 it was 94; at 12 noon it was 92; at 5 p. m. it was 90 mm. Hg.

During these three days with varying doses of strychnin there was practically no effect, even with 1/10 gr., on his pulse or respiration. His pulse showed the greatest effect by a rise of 8 with 1/10 of a grain.

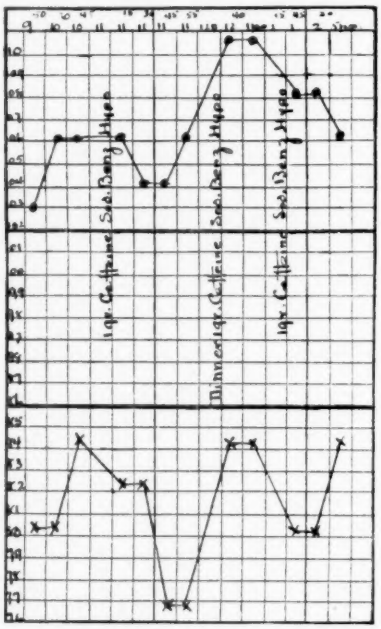

Chart 15.—L. P. Upper curve shows systolic pressure, lower, the pulse-rate under dose of 1 gr. caffein sodium benzoate hypodermically.

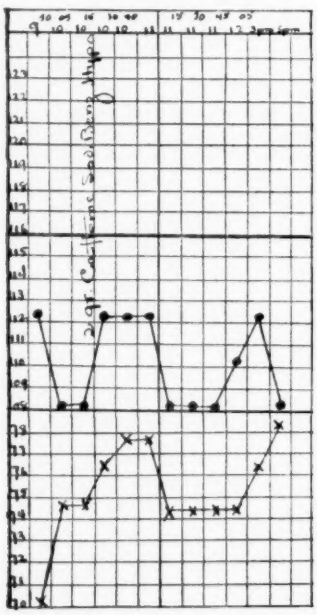

Chart 16.—L. P. Systolic pressure, upper curve, pulse-rate, lower curve, under 2 gr. dose of caffein sodium benzoate hypodermically.

CASE 7.—L. P., aged 14 years, hospital No. 1169, was admitted March 8, 1913.

History.—The patient had chorea five years previously; otherwise had been fairly well. Her present illness had started with pain in the wrist and knees several weeks before admission.

Physical Examination.—The physical examination showed a fairly well developed, but poorly nourished girl. Slight tenderness in wrists. Temperature 100; pulse 90; respiration 20.

Heart: Left border 9.5 cm. from midsternal line in the fourth interspace, 3.5 cm. on the right in the third interspace. Sounds were not very clear, of poor quality, but regular. There was a soft systolic murmur at the apex, transmitted to the base, the axilla and the back. Pulse was very soft, easily compressible and regular.

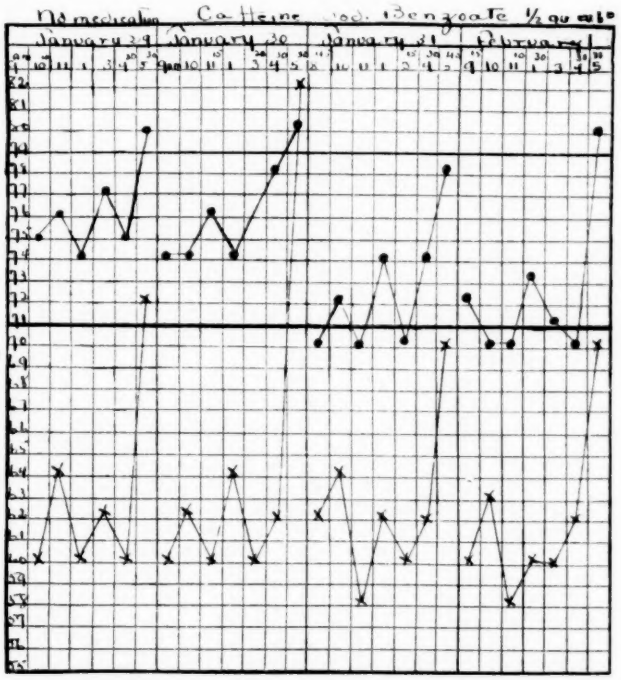

Chart 17.—A. B., aged 8 years. Hospital No. 1136. Myocarditis following pneumonia. Systolic pressure, upper curve, pulse-rate, lower curve, during period of no medication, and of caffein sodium benzoate, 1 gr.. every six hours hypodermically.

Treatment.—She was put to bed and given 25 gr. of aspirin a day for the first three weeks; then was given 5 gr. of aspirin three times a day. During this period she improved markedly. Her heart area diminished by 1 cm. on the left and 0.5 cm. on the right. Cardiac sounds became more clear and pulse stronger. On admission her pulse pressure had been 80 mm. Hg. At the time strychnin was started, April 17, the systolic pressure had ranged between 100 and 110 for the last month, from almost daily observations. On the first day (April 17, see chart) of strychnin observation she was given 1/20 gr. at 9:50 a. m; at 10:50 and at 2:07 p. m. another twentieth by mouth. There was no increase in either the pulse-rate or blood-pressure and no change in her respiration. The next day

she was given 1/10 gr. of strychnin sulphate subcutaneously. Within an hour there was a rise of 12 mm. Hg, with slight twitching around the mouth which lasted for an hour. During this hour her pulse remained within the usual limits and blood-pressure dropped to normal, though she still showed some slight twitching of the mouth. (See chart for April 18.) This patient was also given caffein sodium benzoate. The first day, April 19, she received 1 gr. caffein sodium benzoate at 11 a. m.; another at 12 noon and a third at 1:15 p. m., making 3 grains within a period of three hours without any change in her pulse-rate or blood-pressure. The following day, April 20, she received 2 gr. in one dose without affecting either her pulse or blood-pressure. (See chart for April 19 and 20.)

CASE 8.—A. B., aged 8 years, hospital No. 1136; was admitted to the hospital January 16.

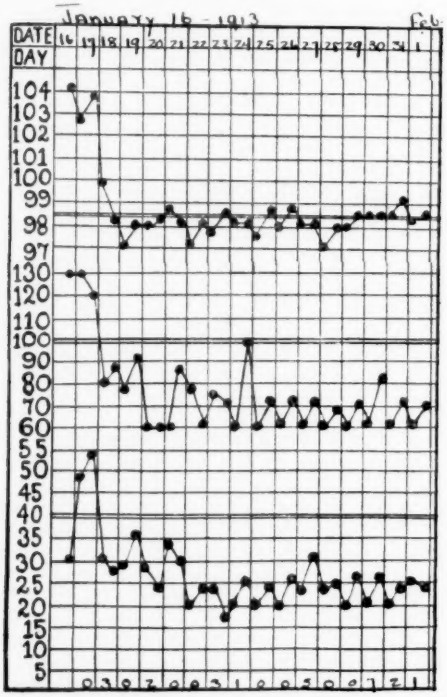

Chart 18.—A. B. Clinical Chart of course of pneumonia.

History.—The patient was a well developed and well nourished boy. He had been in good physical condition until the occurrence of pneumonia, which developed during the week of Jan. 10, 1913. He was admitted with involvement of the left lower lobe. He developed a marked myocarditis during the crisis, which occurred in less than forty-eight hours after his admission, on January 18. He received no medication up to January 29. During this period his temperature was normal, and it remained between 97 F. and normal until January 30, when he was put on caffein sodium benzoate. His pulse had been running between 60 and 70 most of the time, very irregular and small. Respirations were between 20 and 30. During the time that he was on caffein sodium benzoate there was no change in his temperature, pulse or respiration or in his general feelings. His systolic pressure had been running between 75 and 80 mm. Hg previous to starting the caffein. January 30 he received a half grain of caffein sodium benzoate subcutaneously every six hours, starting at 9 a. m.: this was continued for three

days. January 30 at 9 a. m. his systolic pressure was 74 mm. Hg; at 10 a. m. it was 74; at 11:15 it was 76; at 1 p. m. it was 74; at 4:30 p. m. it was 78, and at 5:30 it was 80 mm. Hg.

January 31 at 8:45 a. m., fifteen minutes before he received the fifth dose of caffein sodium benzoate, his systolic pressure was 70 mm. Hg; at 10, an hour after his fifth dose, it was 72; at 11 it was 70; at 1 p. m. it was 74; at 3:15 it was 70; at 4:30 it was 74, and at 5:45 it was 78 mm. Hg.

February 1 at 9:15 a. m., fifteen minutes after the ninth dose, systolic pressure was 72 mm. Hg; at 10 it was 70; at 11:10 it was 70; at 1:30 p. m. it was 73; at 3 it was 71; at 4:30 it was 70; at 5:30 it was 80 mm. Hg. With the rise in systolic pressure there was a corresponding rise in his pulse-rate. There was no change in the heart sounds, which were at all times weak, and there was no change in the irregularity or volume of his pulse as far as could be made out by digital examination.

THE TREATMENT OF GONOCOCCUS VULVOVAGINITIS, WITH FURTHER OBSERVATIONS ON THE VALUE OF THE COMPLEMENT FIXATION TEST IN THE MANAGEMENT OF THIS DISEASE *

GEORGE GILBERT SMITH, M.D.
BOSTON

The gonococcus, in the vulvovaginitis of little girls, infects not only the vulva, but the vaginal mucosa, the cervix, the external os, and not infrequently the urethra. Perrin[1] believes that the gonococcus always infects the cervical canal. He describes the technic which he has used in the examination of about a hundred cases, and says that after wiping the vaginal surface of the cervix free of mucus, one can obtain pus containing the organism from within the cervical canal.

Although some observers have not found the cervical canal involved, all agree that the infection extends well into the vagina. Applications to the vulva alone cannot be expected to cure, since they do not reach the places where the gonococci are. It is on this basic principle that the method of treatment used in the Women's Room of the Genito-Urinary Department of the Massachusetts General Hospital has been developed.

In the very acute stage of vulvovaginitis, local treatment must be introduced with great caution. The vulva, in severe cases, is edematous and perhaps ulcerated; under these conditions the introduction of any instrument or the application of any irritating solution is bound to do harm. For such a case, confinement to bed, regulation of the bowels, much water, a light diet and the frequent bathing of the external genitals with warm boric solution are the measures to be employed.

As the acute condition subsides, and at the outset in less severe cases, the following method has been used by us with considerable success. It can be carried out in the clinic at daily visits, or can be used by any mother of ordinary intelligence who will take the trouble.

TREATMENT OF VAGINITIS

The child is placed on her back on the table, her hips elevated by a douche-pan. The labia are separated and a female soft rubber catheter, size 12 or 14 French, well lubricated, is passed through the opening in

* From the Genito-Urinary Department of the Massachusetts General Hospital.
* Submitted for publication Jan. 9, 1914.

1. Perrin: Rev. méd. de la Suisse romande., Nov. 20, 1911.

the hymen as far as it will go. One is surprised at the depth of the vagina, as the catheter often passes in for an inch and a half.

A fountain syringe is hung about two feet above the patient. The tube should be tipped with the glass part of a medicine dropper, which fits securely into the funnel end of the catheter. The solution selected for this douche, which will be discussed shortly, is allowed to run. From one to two quarts, heated to about 110 in the reservoir, should be used. After this solution has run into the vagina and out again around the catheter, the catheter and glass tip are separated, and through the catheter, which has been held in place all this time, one or two drams of one of the silver salts is injected with a hand syringe. The catheter is then withdrawn, the child holds her thighs close together to prevent the escape of the silver preparation and lies in this position, hips elevated, for at least fifteen minutes. This treatment should be given twice a day, but even if used only once daily, is very effective in checking the discharge. If given by the mother, she is instructed to wash the catheter carefully after each treatment and put it away in a clean towel. She is shown how to insert the catheter, and even in the Out-Patient Department, every woman who has had the desire to try has succeeded in giving the douches without doing any damage.

The solutions used for the douche are sterile water, boric acid solution 2 per cent., sodium bicarbonate a teaspoonful to a quart, or potassium permanganate 1-8,000 or 1-6,000. The purpose of the douche is the removal of mucus preliminary to the application of silver, and the increase of circulation locally through heat. In the more acute stages the blandest solutions are the best, sodium bicarbonate being especially soothing. Later, potassium permanganate, with its slight astringent action, is good, and in the chronic stages, silver nitrate as strong as 1-1,000 may be used.

In choosing a silver salt, one should start with the least irritating preparation. Argyrol 10 per cent. is well borne in the more acute cases; later it can be increased to 20 per cent., or the somewhat more irritant protargol can be used (not stronger than 5 per cent.).

In very chronic or persistent cases it may be expedient to apply tincture of iodin to the cervix. Perrin,[1] in the article mentioned above, advises the use of

Protargol	8 grams
Distilled water	8 grams
Glycerin to	50 grams

An endoscope is passed into the vagina and through this a small swab moistened with this solution is applied to the cervical canal. The same solution is instilled into the urethra. Perrin claims to have

cured about a hundred cases by this method, and in no case did the treatment extend over three weeks.

At one time vaccines were thought to be of great value. We have not used them, nor do we believe that their employment in gonococcus infections of the mucous membrane, whether in the male or in the female, has up to the present given sufficiently satisfactory results to warrant any dependence on them.

URETHRITIS

Urethritis occurs less frequently than in adults. Pollack found this condition in 18 per cent. of 187 cases; Scheuer in fifteen out of thirty-nine patients (both quoted by Norris, "Gonorrhea in Women," page 380).

We have treated this condition by the instillation of 1 or 2 c.c. of 5 per cent. argyrol into the urethra by means of a medicine dropper.

Proctitis we have never noted, nor have we seen any definite cases of extension of the infection above the cervix. These complications do occur and must be borne in mind.

DURATION OF TREATMENT

The treatment should be carried on for at least a month after all symptoms have disappeared. One must remember, however, that occasionally a discharge may be kept up by too persistent treatment. After treatment is stopped the case should be seen twice the first week, then once a week for three weeks, and even after that should be followed for several months. Smears should be taken from the depths of the vagina by inserting a small wire swab. When the disease is cured the smear should consist of mucus and epithelial cells, with practically no pus cells, and of course the diplococcus of Niesser should not appear.

Should children with gonococcus vulvovaginitis be allowed to attend school? We have believed that if their condition were made known to the teacher, it could be so arranged that they might not have to use the common toilet. This danger removed, they are less likely to be a focus of infection while in school than when playing untended on the street.

THE QUESTION OF CURE

In a previous paper[2] on "The Complement Fixation Test in the Management of Gonococcus Vulvovaginitis," we stated the evidence for our belief that this disease is curable. The contrary opinion is held by not a few, who maintain that although the infection may have periods of quiescence it is more than likely to reappear sooner or later. Edith

2. Smith, G. G.: Am. Jour. Dis. Child., 1913, v, 313.

Spaulding,[3] in her very comprehensive paper on this subject, states that "the disease may extend over many years, during which time there may be many recurrences, and that the periods of latency may at least be as long as eighteen months." She also says that "the most efficient treatment does not insure a permanent cure." With such pessimistic conclusions we cannot agree. We cannot believe that the characteristics or clinical manifestations of the gonococcus in the vagina differ very greatly from its characteristics or clinical manifestations in the male urethra, and certainly in the latter situation the disease is curable. Wherever glands or crypts occur, as they do both in the cervical canal and in the male urethra, the infection will pursue an irregular course, at times almost disappearing, and again breaking out in profuse discharge; in deciding on the question of cure, therefore, this peculiarity of the gonococcus must be considered, and no case considered well until considerable time has passed during which there is no evidence of infection. This period in vulvovaginitis we would arbitrarily set at three months, although we believe the patient should be seen at intervals after that.

If after three or more months, during which frequent smears from the depths of the vagina have contained no pus and all other signs of infection have disappeared, a discharge containing gonococci recurs, we believe that this attack is frequently a reinfection, very possibly from the same source as the first. It is worth while to consider the likelihood of such a reinfection, and to investigate the home conditions surrounding the patient. The Children's Department at the Massachusetts General Hospital maintains a social worker who has been of great assistance in this way.

With this view as to reinfection Spaulding disagrees. She argues that of the twenty-six cases in her series, twenty-two had second attacks; and that some children who were infected in hospitals had recurrences after going home, whereas their sisters were not infected. The answer to these arguments is that in the majority of her cases the test of cure was insufficient. Thus in ten of the twenty-two recurrences, two months or less was the greatest interval between attacks. In one of the two cases in which vulvovaginitis recurred after two years of apparent health, Spaulding admits the possibility of a reinfection from other members of the family who were found to have gonorrhea.

Another explanation of attacks of vulvovaginitis following the original infection after a considerable period of health is that they are not due to the gonococcus at all, but to the other organisms which find that several weeks or months of suppuration have made the mucosa of the vagina a favorable ground for development. It is well known that

3. Spaulding: Am. Jour. Dis. Child., 1913, v, 248.

non-specific posterior urethritis occurs with vastly more frequency in men who have had a gonococcus infection at some preceding time, and it is our belief that for similar reasons non-specific vulvovaginitis is likely to occur in girls who have been infected by the gonococcus. The discharge in such cases looks like chalk and water, resembling in appearance the leukorrheal discharge of women. Under the microscope it shows epithelial cells in abundance and comparatively few pus cells. In two or three of our cases a very large Gram-positive bacillus was present. Silver nitrate douches in strength ranging from 1-5,000 to 1-1,000 have given the best results with this non-specific vaginitis.

Another type of vulvovaginitis not due to the gonococcus is characterized by irritation of the skin about the genitals and on the inner surface of the thighs; the discharge is scanty in amount, watery in consistency, and appears too slight for the extent of the dermatitis. Smears from the vagina show no pus, and it is our suspicion that the irritation is due to frequent masturbation.

As further evidence on the question whether gonococcus vulvovaginitis can be cured with any regularity, and also on the value of the complement fixation test in this connection, we wish to present the histories carried down to date of sixteen of the twenty-five cases reported in our earlier paper. The other nine we have been unable to find. In all these cases but one smears were taken from the depths of the vagina, stained by Gram's method and examined by us. The presence or absence of discharge was noted, in most cases a history was obtained, and in all the blood was submitted to Dr. James Homer Wright, pathologist at the Massachusetts General Hospital, for the complement fixation test.

The first ten cases are those which had negative bloods at the time the last paper was written (on or before Dec. 7, 1912). The duration of treatment of these cases in our clinic varied from a few weeks to two years. In some of them (Case 18 especially) the treatment was merely instituted by us and carried out entirely by the mother at home. Most of the cases came for treatment once or twice a week, and between visits were treated at home.

CASE REPORTS

CASE 1.—O. P. D., 177,098, M. McC. Infected August, 1911. Nov. 7, 1912. Some redness and smegma about genitalia. Smear: no gonococci. Blood negative. Nov. 1, 1913. Mother has seen no discharge since last visit. Dermatitis of labia majora and adjacent skin. No discharge; no redness of introitus. Smear: epithelium; rare leukocyte; a few Gram-positive cocci; no gonococci seen. Blood weakly positive.

CASE 2.—O. P. D., 119,061, G. G. Infected May, 1911. Feb. 3, 1912. No discharge. Smear negative. Blood negative. Nov. 1, 1913. Genitalia appear normal. No discharge. Has a suppurating gland in neck. Smear from vagina:

small number of pus cells, one of which contains Gram-negative cocci, not morphologically gonococci. Blood weakly positive.

CASE 3.—O. P. D., 186,609, H. C. Raped November, 1911. January, 1912. Gonococci present. March 11, 1912. Blood weakly positive. June 10, 1912. Blood negative. Nov. 22, 1912. No discharge noted by mother for over six months, except for three days in July, 1912; stopped when she gave the child a few douches. Smear: no pus; no gonococci. Blood negative. Dec. 5, 1913. Has had no discharge for seventeen months. Genitalia appear normal. Smear: epithelial cells, mucus, a few leukocytes. No gonococci seen. Blood weakly positive.

CASE 4.—O. P. D., 191,815, A. O. Raped November, 1911. Treated at Long Island Hospital (in-patient) from then until April 2, 1912. April 10, 1912. No discharge. Smear negative. Blood negative. Sept. 4, 1913. Has had no discharge for at least seventeen months. Genitalia normal. Not enough discharge for a smear. Blood weakly positive.

CASE 5.—O. P. D., 139,370, E. C. Infected in 1906. Gonococci present in 1910. April, 1912, blood positive. Nov. 23, 1912. Blood suspicious. Dec. 7, 1912. Blood negative. Smear: no pus, no gonococci. Sept. 4, 1913. No discharge for at least ten months, she says. Examination shows some leukorrhea of whitish color. Introitus slightly reddened. Smear: epithelium, no pus cells. A very large Gram-positive bacillus in large quantities. No gonococci seen. Blood weakly positive.

CASE 6.—O. P. D., 51,067, A. R. Infected June, 1911. Nov. 14, 1912. Mother has seen no discharge for a year. Blood weakly positive. Dec. 7, 1912. Blood negative. Smear: no pus, no gonococci. Oct. 30, 1913. No discharge for two years. No redness of genitalia. Smear: epithelium and mucus, no pus. Many Gram-negative bacilli. No gonococci. Blood negative. Dec. 6, 1913. Returns with complaint of brief sharp pains in lower abdomen. Very slight tenderness on deep suprapubic pressure. Rectal examination shows nothing abnormal. Considerable leukorrhea. Smear: epithelium, some pus. Large Gram-positive bacillus in great abundance. No gonococci seen.

CASE 7.—O. P. D., 159,570, L. S. Infected 1907. June 15, 1912. Labia red, no discharge. Blood negative. Nov. 22, 1913. Has had some discharge on clothes occasionally; some during past week. "Is much better than she was once." Two months ago had watery red discharge (mother's statement). Labia very red and irritated. Thin watery discharge. Smear: epithelium, a few pus cells. Gram-negative diplococci, larger than gonococci and not intracellular. Blood suspicious.

CASE 8.—O. P. D., 160,329, L. S. Sister of Case 7. Infected in 1910. June 11, 1912. Mother sees discharge at times. Blood negative. Nov. 22, 1913. No discharge for over a year. Genitalia normal. Smear: epithelium, no pus, no gonococci. Blood negative.

CASE 9.—O. P. D., 163,160, B. M. Infected October, 1910. Dec. 2, 1912. No discharge for eighteen months. Nov. 22, 1913. Some whitish discharge at times (mother's statement). None seen at examination. No redness. Smear: epithelium and mucus; no pus, no gonococci seen. Blood negative.

CASE 10.—O. P. D., 183,401, B. E. Infected November, 1911. March 9, 1912. No discharge. Blood negative. Dec. 13, 1912. No discharge now or since last visit. Smear: rare pus cell, no gonococci. Not seen since.

CASE 11.—Last test Nov. 23, 1912, weakly positive. Not seen since.

CASE 12.—Last test Nov. 21, 1912, suspicious. Not seen since.

CASE 13.—O. P. D., 197,035, I. K. Infected June, 1910. Oct. 19, 1912. Blood weakly positive. Nov. 23, 1912. Slight redness of vestibule. No discharge. Smear: no pus, no gonococci. Blood weakly positive. Feb. 15, 1913. Blood negative. Oct. 25, 1913. Has had no discharge for a year. No discharge. No

redness. Smear: epithelium, no pus, no gonococci. Very few bacteria in smear. Blood weakly positive.

CASE 14.—Last test May, 1912, positive. Not seen since.

CASE 15.—O. P. D., 177,097, G. McC. Sister to Case 1. Aug. 28, 1911. Gonococci present in smears. Nov. 14, 1912. Occasional discharge (mother's statement). No gonococci in smear. Nov. 1, 1913. Dermatitis of external genitalia. No discharge or redness of introitus. Smear: epithelium, rare pus cell, a few Gram-positive cocci. No gonococci. Blood weakly positive.

CASE 16.—Last test Nov. 18, 1912, positive. Not seen since.

CASE 17.—O. P. D., 141,770, I. C. Sister to Case 5. Infected January, 1910. May, 1912. Blood negative. Oct. 19, 1912. Discharge, which has been absent for six months, began again. Blood weakly positive. Sept. 4, 1913. Has discharge at times (sister's statement). Examination: genitalia normal. Smear: some mucus and epithelium. No pus. No gonococci seen. Blood weakly positive. Nov. 18, 1913. Blood negative.

CASE 18.—O. P. D., 143,269, K. C. Infected September, 1912. Nov. 23, 1912. Smear: no gonococci. Blood strongly positive. Nov. 1, 1913. Has had no signs of discharge for over a year (mother's statement). Genitalia normal. No discharge. Smear: epithelium, mucus; no pus, no gonococci. Blood moderately positive.

CASE 19.—O. P. D., 196,190, S. J. Infected June, 1912. Dec. 7, 1912. Some discharge. Smear: gonococci present. Blood suspicious. July 22, 1913. Blood negative. Oct. 25, 1913. No discharge for nine months. Genitalia normal. Smear: epithelium; no pus, many bacteria but no gonococci seen. Blood negative.

CASE 20.—Last test December, 1912, positive. Not seen since.

CASE 21.—Last test Nov. 23, 1911, weakly positive; not seen since.

CASE 22.—O. P. D., 196,813, B. A. Infected May, 1912. Dec. 7, 1912. Some discharge. Gonococci present in smear. Blood weakly positive. March 15, 1913. Blood weakly positive. Oct. 25, 1913. Blood negative. Has had douche to-day. Nov. 1, 1912. Some discharge. Smear: much pus, many diplococci, chiefly Gram-positive. A few atypical Gram-negative diplococci. These declared not to have the morphology of the gonococcus by Dr. J H. Wright.

CASE 24.—O. P. D., 149,629, S. G. Sister to Case 2. Infected January, 1911. December, 1911. Gonococci present in smear. February, 1912. No discharge. Blood positive. Nov. 1, 1913. Slight amount of thin white discharge. Smear: epithelium, mucus, no pus. Many Gram-positive cocci. No gonococci seen. Blood moderately positive.

CASE 25.—Last test May, 1912, weakly positive. Not seen since.

DISCUSSION

Of the ten cases which had negative bloods and apparent cures one year ago, nine returned this year for examination. Of these only three now have negative blood tests; six are weakly positive or suspicious. Of these six, five give every sign of cure from the clinical point of view. All five had negative blood tests one year ago and not one has had any discharge since; yet the blood test now is weakly positive!

The finding of a weakly positive test in these cases which gave a negative test last year and have shown no signs of infection since, appears to be due to the greater sensitiveness of the test which has developed with further use. The belief is held by Dr. Wright and other workers that a slight inhibition of hemolysis (as seen in weakly positive tests) may be due to a so-called "group reaction"—that is,

gonoccus antigen will combine to a slight degree with the antibodies due to allied micrococci. Irons,[4] states that occasionally in adults, and more frequently in children, a fairly positive reaction occurs in persons who have never had gonorrhea.

We have had under observation a girl of 18 who has been clinically cured of gonorrhea for over two years. She lately married and has borne a child, without the slightest sign of infectiousness or even discharge. Her blood is persistently weakly positive.

It is our belief, therefore, that in the determination of a cure a weakly positive test cannot be considered evidence that infection is still present. A negative test, on the other hand, appears to be proof of its absence.

Of the seven cases which had weakly positive blood tests a year ago, five are now clinically well. Of these, two have negative bloods, one a moderately positive and two weakly positive. The other two show some vaginitis in which, however, gonococci could not be found.

From the facts as shown in these sixteen cases, can we say that gonococcus vulvovaginitis is a curable disease?

It seems to us that in 50 per cent. of these cases (Nos. 1, 3, 4, 8, 9, 13, 17, 19) the evidence of cure would satisfy the most skeptical. Of the remaining 50 per cent. (Nos. 2, 5, 6, 7, 15, 18, 22, 24) no one seems to be still definitely gonorrheal. Case 2 is not cited as a cure because the patient has no definite history of freedom from discharge. Case 5 has a leukorrhea, which contains a large bacillus in almost pure culture. Case 6 would have been given a clean record on Oct. 30, 1913, but the leukorrhea which has started up since then spoils her record, although it does not appear to be due to the gonococcus. Cases 7, 15 and 22 may well be due to the gonococcus, although that organism was not found. Cases 18 and 24 we rule out because of a moderately positive blood test, although in every other respect Case 18 appears to be a cure.

The study of these cases, few though they are, warrants, we believe, a fairly optimistic feeling as regards the prognosis in this disease. Long and persistent treatment, frequent and severe tests of its probable cure, are necessary to complete success. We believe that in the great majority of cases of vulvovaginitis efficient treatment will insure the destruction of the gonococcus; a small proportion of such cases, however, will be liable to attacks of non-gonococcal inflammation for some time.

We wish to acknowledge our indebtedness to Dr. James Homer Wright, under whose direction the complement fixation tests were done, and to Miss Clara M. Welsh, social worker of the Children's Department of the Massachusetts General Hospital, through whose efforts the children mentioned in this paper were brought back to the clinic for examination.

4. Irons: Jour. Infect. Dis., July, 1912, p. 77.

NOTES ON A PROBABLE RELATION BETWEEN ENLARGEMENT OF THE THYMUS BODY, LYMPHATISM AND SHOCK*

H. C. CLARK, M.D.
ANCON, CANAL ZONE

The function of the thymus gland is still, perhaps, little understood. Of late years, however, more or less general attention has been given it by clinical as well as experimental observers and a certain amount of circumstantial evidence is being recorded against it. At times the gland has assumed a medicolegal importance in instances of sudden death. For years it has been known that in many cases of thyroid disease the thymus becomes greatly enlarged. In marantic children it frequently is found in a marked atrophic state. In certain instances there is a failure in involution and an active gland persists, yet no one can fully explain whether its persistence in adult life or its enlargement when associated with goiter is one of Nature's safeguards in preserving the equilibrium of the body or whether there is primary disease in the thymus. No one knows what the true relation of the thymus gland may be to the lymphadenoid system and yet many observations have been recorded of changes which seem to have progressed hand in hand in these structures. No tangible relation seems to exist between infection and the thymus-lymphadenoid system. Many circumstances lead to the idea that this system is under highly specialized nervous control, as for instance, in children possessing a "lymphatic constitution" sudden fear or anger may provoke an attack of "thymic asthma." The administration of an anesthetic would seem, from the evidence accumulated, to be a sufficiently strong exciting factor to throw this system into dangerous activity.

Because it is so difficult to point out the rôle played by these structures in disease and death, it seems advisable to offer what little evidence may have been collected at Ancon showing the apparent inter-relation of the thymus and lymphadenoid system in various forms of shock.

In December, 1911, it became necessary to investigate the cause of death in an American child which died suddenly in Ancon Hospital from asphyxiation.

---

* Published with permission of the Chief Sanitary Officer, Colonel W. C. Gorgas, U. S. A.

* From the Board of Health Laboratories, Ancon, C. Z.

CASE REPORT

History.[1]—The patient was a female child, aged 15 months, born on the Canal Zone of American parents. Admitted to Ancon Hospital Dec. 6, 1911. There was a history of cough lasting seven days, with, at times, blood-tinged mucous sputum. No whoop. Patient was brought to the hospital by its parents who were greatly excited over recent "spells of suffocation" through which the child had passed. At the time of admission the child appeared perfectly well aside from a moderate degree of anemia. Doubt was therefore cast, for the time being, on the severity of the conditions related by the parents. Instructions were given the nurses and physicians in charge to watch carefully for any manifestation of the above symptoms.

Physical Examination.—The physical examination of the thorax revealed a few scattered râles but in all other respects the lungs were quite normal. Head, neck and abdominal examination revealed no variations from the normal. The patient played and talked as any other child until some hours after admission, when it was seized with a paroxysm of choking and coughing. The nurse was compelled to resort to artificial respiration, and strychnin was given as soon as a physician could arrive. So short was the attack that the resident physician in charge failed to arrive in time to see the paroxysm at its height. The child regained and maintained its normal condition until 2:30 p. m., December 7, when again it was seized in a similar manner and died before intubation could be performed. The temperature at no time had exceeded the range of 99 to 100.5 F., the latter being terminal. The pulse during the paroxysm was 128 and respirations recorded prior to seizure, about 40. The child is said to have died a typical death by suffocation.

No examination of blood, urine, or stool had been entered on the history sheets. It was sent to the laboratory with the entry of "sudden death of undetermined origin."

Necropsy.—Anatomic findings are entered under the number A-2860. The necropsy was performed one and a half hours after death.

The child appeared well nourished but was pallid. The skin had the appearance of a skin suddenly exposed to cold ("goose-flesh") but more marked than is usually seen.

Internal examination absolutely failed to associate any organic disease or infectious process. The cranial cavity was quite normal but for injection of vessels. The throat showed no evidence of diphtheria or any allied disease and no visible laryngeal obstruction from mechanical causes. The tonsils and lymphoid follicles about the base of the tongue were, however, greatly enlarged, apparently a hyperplasia.

Thorax: A huge thymus of anomalous shape and thickness extending in a bicornuate fashion above the manubrium and well down into thorax to about the fourth interspace was found. It was quite thick beneath the manubrium and closely applied over the large vessels and pericardium. Its dimensions were 13 cm. x 6.5 cm. x 15 mm. and weighed 35 gm. The trachea was severed a few centimeters above the edge of the sternum and then it was found that the thickest portion of the gland was partially obstructing the trachea at the level of the manubrium. No doubt the lumen of the innominate vein had been quite seriously encroached on. The lungs were emphysematous and a few punctate hemorrhages were found in the pleura. The only other pulmonary lesions were a few small patches of capillary bronchitis and tiny little alternating areas of elevation and depression over the visceral pleura simulating areas of atelectasis and emphysema. All thoracic glands appeared usually large, fleshy and pale. The heart had a few punctate hemorrhages in the epicardium and a moderate dilatation of the right side.

---

1. The clinical notes were kindly given me by Dr. W. E. Deeks, chief of Medical Clinic at Ancon.

Abdominal Cavity: Absolutely normal aside from the same glandular enlargement mentioned above and a very marked degree of hyperplasia (even though it be a child's body) of all follicles and Peyer's patches in the intestine. Marked enlargement of the malpighian bodies of the spleen. The rib and femur marrow showed a marked hyperplasia. The investigation failed to reveal an infection, or an organic disease, barring the acceptance of "lymphatism" as a pathologic entity. Wassermann test on the post-mortem blood was done by Dr. L. B. Bates and found negative. The microscope failed to indicate anything further than an intense lymphadenoid hyperplasia. There was intense hyperplasia of all germinal follicles.

DISCUSSION

This case to my mind represents a death due to lymphatism without a recognizable associated disease—a pure case of status lymphaticus resulting in death.

The findings would lead one to believe that the unusual thickness of the upper extremity of this gland had brought about death by pressure on both the trachea and the large vessels at the base of the heart, especially the innominate vein, some exciting agent causing a degree of congestion and edema of the abnormal thymus, and a corresponding embarrassment of the innominate vein and heart followed by tracheal stenosis.

Fetterolf[2] and Gittings, in working out some anatomic features of the child's thorax, used bodies injected with 10 per cent. formaldehyd solution which were afterward frozen and then studied in serial cross sections. This seems an admirable way to study the true relations. They point out the following facts in relation to this structure:

The thymus body is a structure of considerable size and extent, reaching well beyond the borders of the sternum and separating widely the upper lobes of the lungs. Posteriorly, at least in the first few weeks of life, it extends in places to the trachea. The upper median portion of the gland is deeply indented posteriorly by the left innominate vein, which lies immediately on the anterior surface of the trachea.

They conclude that where the gland does extend to the trachea it would seem that sudden death, at this period due to respiratory failure, might be caused by even moderate increase in the size of the gland. Delay in normal involution of the body would extend the danger period. They also feel that when the size and distensibility of this vessel and the softness and small caliber of the trachea are taken into consideration that a possible mechanical explanation of the sudden death which occurs in the young during anesthesia may be explained. It was also stated by them that any factor which would embarrass the right heart would dilate this vein, which, being braced on its anterior surface by the thymus, would be forced to extend itself posteriorly, resulting in a greater or less degree of tracheal compression. If compression were great, sudden death would follow; if of slight degree the amount of asphyxia caused would tend to raise pressure and cause still further dilatation of the innominate vein, thereby creating a vicious circle which would result in death.

2. Fetterolf and Gittings: Am. Jour. Dis. Child., 1911, i, 6.

Dudgeon's[3] observations on the weight of the gland have been compiled from a large series of records and are perhaps the most valuable. He states that the thymus varies enormously in weight according to the health and age of the child. The average weight of the gland after birth up to the age of 2 years is 7 to 10 gm. A gland of 25 to 30 gm. is considerably enlarged. Dudgeon collected sixteen cases of "sudden death," "found dead" and "lymphatism." Seven of these closely resembled status lymphaticus and four fulfilled all requirements.

Close attention has always been given the gland in performing autopsies on marantic or rickety subjects, finding them as a rule greatly atrophied and weighing from 1 to 5 gm. in children 6 to 18 months of age. The case just described, however, awakened an interest in its other possibilities, and since then a series of 500 cases of all ages have come to hand and in each case the gland was given rather more than the usual attention. The general belief is that the body seldom persists after the fifteenth year. This has not been the experience at Ancon. Almost without exception, regardless of the age or race, some thin remnants of thymic tissue can be found. It must be admitted, however, that few of them contained much cellular material. Many of the deaths at Ancon have been associated with some form of trauma and shock and as many of these victims have been young adults the findings have sometimes proved interesting.

THE THYMUS FINDINGS IN FIVE HUNDRED NECROPSIES

In a series of 500 consecutive autopsies, observations on the thymus and the lymphadenoid system were made, and as a working index all cases exhibiting a thymus gland weighing 10 gm. or more were taken for study. Thirty-five cases were found as tabulated.

A glance at the table will show what, arbitrarily, may be called variations. General hyperplasia of the lymphadenoid system is expressed by one + sign; a double ++ indicates marked hyperplasia, and a triple +++, cases showing an extreme degree of the same change.

It should be stated again that most all of the cases coming to autopsy at Ancon are young black or mixed race adult males; therefore the lack of variation in race, age and sex found in the series is explained and comparison is not possible with most other statistics. In those cases not accompanied by traumatic influence the thymus ranged in weight from 10 to 15 grams, and were chiefly persistent remnants enlarged by edema and congestion, except for the possible effects of the toxins of the pneumococcus and the malarial parasite. There have been several cases of

3. Dudgeon, Leonard S.: A Contribution to the Pathology of the Thymus Gland, Jour. Path. and Bact., 1905, x, No. 2, p. 173.

malaria in children examined and only five of them have fallen in this group taken to represent a pathological variation. A-2983, A-3046 and A-3158 showed a very marked increase in size and even when the ages, 7 years and 4 years, are taken into consideration, there would seem to remain a condition out of proportion to the changes usually accompany-

TABLE 1.—THYMUS FINDINGS IN THIRTY-FIVE CASES

Autopsy No.	Race	Age, Yrs.	Sex	Cause of Death	Thymus weight, Gm.	Lymphoid Hyperplasia General
2860	U. S. A.	1¼	F	Lymphatism	35	+++
2861	Black	13	M	R. R. traumatism	40	+++
2863	Black	35	M	Lobar pneumonia	10	+
2882	Spain	29	M	Brain embolus	30	+
2887	?	38	M	R. R. traumatism	22	+
2888	Mixed	24	M	Homicide	20	+
2889	Mixed	23	M	R. R. traumatism	15	+
2893	Black	24	M	R. R. traumatism	25	+
2894	Black	30	M	Drowning	15	+
2937	Black	24	M	Cerebral thrombosis	12	+
2948	Black	17	M	Anesthesia and tuberc.	47	+++
2955	Black	20	M	Malaria	10	+
2958	Hindu	30	M	Trauma—abdom. blow.	15	+
2983	Spain	7	M	Malaria	25	+++
2985	Black	20	M	Lobar pneumonia	20	+
2986	Mixed	22	M	Typhoid	30	++
2988	Black	15	M	Chronic nephritis	10	+
3013	Black	20	M	R. R. traumatism	30	+++
3015	Spain	25	M	Infection unknown	25	+
3039	Mixed	5/12	F	Pyemia pneumococcus	12	*
3046	Black	4	M	Malaria	17	+++
3064	Mixed	2/12	F	Trauma by fall	45	+++
3072	Mixed	25	M	Lobar pneumonia	25	+
3082	Black	22	M	R. R. traumatism	13	+
3087	Black	27	F	Eclampsia	10	?
3097	Spain	6	M	Malaria	20	++
3102	Spain	5	M	Malaria	20	++
3114	Black	22	M	R. R. traumatism	21	++
3158	Black	7	F	Malaria	32	+++
3279	Black	25	M	Alcoholism (?)	31	*++++
3320	Black	28	M	Anesthesia operation and R. R. traumatism	15	++
3334	Mixed	9	M	Sarcomatosis	15	++
3338	Mixed	23	M	Lobar pneumonia	15	+
3339	Black	15	M	R. R. traumatism	52	+++
3351	Black	20	M	R. R. traumatism	25	++

* Acute thymitis.
† Hemorrhages.

ing malaria in the lymphadenoid system of children of this age. One child was free from malarial parasites in the peripheral blood and the prognosis considered bright, when an unexpected sudden death occurred. In two of the cases of pneumonia, A-2985 and A-3338, the small amount of involvement would not ordinarily have offered a fatal prognosis in

young men of 20 years and 23 years, respectively, even though it be granted that the Negro has little resistance against the pneumococcus.

The most interesting group was that including the deaths due to violence. There were thirty-five deaths in the 500 cases due to traumatic influences. A few were not submitted to a complete autopsy but in thirty instances records were made. Reference to the table will show that in thirteen of these cases this peculiar lymphatic state was found in the victims of railroad traumatism, homicide, drowning, fall or blows. In A-2861, A-2889, A-3013, A-3114 and A-3339, the injuries sustained would not as a rule have indicated a very doubtful prognosis.

Such conditions as cerebral embolism and thrombosis, eclampsia during labor, surgical shock, etc., have occasionally shown this condition.

A-2948 represents a 17-year-old black male who had been operated on for cervical lymphadenitis and died while under the anesthetic. A widely disseminated tuberculous condition was found and a large mass of glands partially obstructed the right bronchus. A huge thymus offered some degree of stenosis to the trachea.

Three cases of malaria, two of pneumonia and five due to trauma, which, from a clinical view-point, would ordinarily offer a fair prognosis, are thus found in fatal association and presenting to a marked degree this peculiar lymphatic state.

These notes have been presented to invite further attention to the probable relation this lymphatic state may bear to shock.

Is this state a defensive effort on the part of Nature to compensate for some weakness in another system? May it be the result of violence or disease, or a defensive reaction of some kind? Can it be a primary variation from the normal in the thymus and lymphadenoid system? It is not the desire to look on this feature as frequently the direct cause of death but to point out its presence and apparent association in certain cases in the production of shock. In ten of the tabulated cases, this peculiar lymphatic state certainly seems to indicate a pathological condition (primary weakness) in the lymphadenoid system which contributed to a fatal issue.

To recapitulate: Case A-2860 represents a fatal case of lymphatism without associated disease, and in which the cause of death was unassociated with any known form of external violence.

Some individuals seem to present an abnormal hyperplasia of the thymus and lymphadenoid system or these structures fail to undergo the proper degree of involution when adult life is reached, the result being for some unknown reason a lessened resistance to disease and shock, especially the latter.

Thanks are due Dr. S. T. Darling for many valuable suggestions in preparing this paper.

RULES FOR CALCULATING THE APPROXIMATE COMPOSITION OF MILK FROM THE SPECIFIC GRAVITY AND PERCENTAGE OF FAT AS DETERMINED BY THE BABCOCK METHOD

H. I. BOWDITCH, M.D., AND A. W. BOSWORTH, A.M.
BOSTON GENEVA, N. Y.

In a previous paper[1] we gave a rule for finding the percentage of total protein in milk from the percentage of casein. As printed it reads: "The percentage of casein multiplied by 1.4 will give the percentage of total protein . . ." This is incorrect and should read: "The percentage of casein multiplied by 1¼ will give the percentage of total protein . . ."

This rule is based on the figures given in the accompanying table:

TABLE 1.—DEMONSTRATING RULE THAT CASEIN × 1¼ = TOTAL PROTEIN

Casein Found	Total Protein Found	Total Protein* / Casein	Casein Found	Total Protein Found	Total Protein* / Casein
2.54	3.16	1.244	3.01	3.79	1.252
2.42	2.99	1.235	3.13	4.04	1.290
2.46	3.04	1.235	2.29	2.81	1.227
2.52	3.13	1.242	2.34	3.02	1.290
2.61	3.25	1.245	2.47	3.24	1.301
2.68	3.33	1.242	2.43	3.07	1.262
2.71	3.40	1.245	2.39	3.02	1.262
2.80	3.47	1.239	2.55	3.20	1.255
2.90	3.57	1.231	2.81	3.55	1.262
	Average.....				1.252

We also wish to call attention to the fact that the approximate composition of milk may be found by a few simple calculations from the lactometer reading and the percentage of fat as determined by the Babcock method. The rules for these calculations are as follows:

1. Total solids:
   L/4 + (1.2 × F) = total solids.
2. Solids not fat:
   L/4 + (0.2 × F) = solids not fat.
3. Casein:
   (F — 3) × 0.4 + 2.1 = per cent. of casein.
4. Total proteins:
   (a) (F — 3) × 0.4 + 2.8 = per cent. total proteins.
   (b) Casein × 1¼ = per cent. total proteins.

1. AM. JOUR. DIS. CHILD., 1913, vi, 394.

* The figures under this heading were obtained by dividing the per cent. of total protein by the per cent. of casein.

5. Sugar:
   (a) Total solids — (F + total protein + 0.9) = per cent. sugar.
   (b) Solids not fat — (total protein + 0.9) = per cent. sugar.
   L = the reading of the Quevenne lactometer.
   F = the per cent. of fat as determined by the Babcock method.

In rules 5 (*a*) and 5 (*b*) the 0.9 is introduced in order to compensate for the ash and organic substances not otherwise accounted for (citrates, etc.).

Rules 1, 2, 3 and 4 (*a*) are taken from "The Science and Practice of Cheese Making," by Van Slyke and Publow.

The accuracy of these rules is shown by the figures given in Table 2.

TABLE 2.—APPROXIMATE COMPOSITION OF MILK CALCULATED FROM LACTOMETER READINGS AND BABCOCK TEST

	Solids	Fat	Casein	Total Protein	Sugar, Ash, Etc.	Sugar
Found....	11.98	3.43	2.29	2.81	5.74	
Calculated	.....	....	2.27	2.85	5.70	4.80
Found....	12.43	3.58	2.34	3.02	5.83	
Calculated	.....	....	2.33	2.91	5.94	5.04
Found....	12.64	3.64	2.47	3.24	5.76	
Calculated	.....	....	2.36	2.95	6.05	5.15
Found....	12.52	3.62	2.43	3.07	5.83	
Calculated	.....	....	2.35	2.94	5.96	5.06
Found....	12.65	3.84	2.39	3.02	5.79	
Calculated	.....	....	2.44	3.03	5.78	4.88
Found....	12.86	3.98	2.55	3.20	5.68	
Calculated	.....	....	2.49	3.11	5.77	4.87
Found....	13.50	4.23	2.81	3.55	5.72	
Calculated	.....	....	2.59	3.24	6.03	5.13

PROGRESS IN PEDIATRICS

INDEX TO CURRENT PEDIATRIC LITERATURE

ANATOMY AND PHYSIOLOGY

Stomach Digestion in Infants.—H. Davidsohn.
Ztschr. f. Kinderh., Berlin, Jan. 31, 1913.

Weight First Twelve Days After Birth.—G. Benestad.
Arch. f. Gynäk., Jan. 17, 1914.

HYGIENE

Infant Mortality, Ancient and Modern.—L. E. Holt.
Arch. Pediat., December, 1913.

Heat and Infant Mortality.—J. W. Schereschewsky.
Arch. Pediat., December, 1913.

Medical Supervision of Child Between Infancy and School Age.—H. R. L. Shaw.
Arch. Pediat., December, 1913.

Schoolchildren, Physical Condition of Retarded.—S. J. Baker.
Med. Rec., New York, Jan. 10, 1913.

BACTERIOLOGY AND PATHOLOGY

Acardius Monster.—R. Klotz.
Arch. f. Gynäk., Feb. 7, 1914.

Blood-Cultures, A Simple Technic for, in Children.—Jesse R. Gerstley.
Jour. Am. Med. Assn., Jan. 31, 1914.

Cancerous Meningitis; Twenty-four Cases.—Humbert and Alexieff.
Revue de méd., December, 1913.

Cancer of the Scalp, Inoperable, on Boy of 14. Recurrence after Two Operations since Age of 6.—Etienne and Aimes.
Ann. de méd. et chir. inf., Jan. 1, 1914.

Neck, Congenital Malformation of the.—Charles A. Parker.
Jour. Am. Med. Assn., Feb. 7, 1914.

Renal Infantilism.—R. Miller and L. Parsons.
Clin. Jour., London, Dec. 31, 1913.

Status Lymphaticus.—C. McNeil.
Edinburgh Med. Jour., January, 1914.

Toxemias, Periodic Explosive.—B. B. Crohn.
New York Med. Jour., January 17, 1913.

Xiphopagus.—G. Le Filliatre.
Bull. de l'acad. de méd., Jan. 6, 1914.

METABOLISM AND NUTRITION

Albumin-Milk in Infant Feeding under Pathologic Conditions.—A. Lichtenstein.
Hygiea, Stockholm, January, 1914.

Acid Intoxication in Infants, Unusual Type of.—I. A. Abt.
Am. Jour. Med. Sc., January, 1913.

Acid Production and Nervous Over-Excitablity.—H. Elias.
Wien. klin. Wchnschr., Jan. 8, 1914.

Autotoxemia and Acidosis.—T. G. Croft.
S. C. Med. Assn. Jour., January, 1914.

Diet, Dystrophy and Diarrhea in Infants.—W. F. Litchfield.
Australasian Med. Gaz., Dec. 20, 1913.

Feeding, Measured, for Older Children.—W. R. P. Emerson.
Boston Med. and Surg. Jour., Jan. 15, 1913.

Infant-Feeding from New Viewpoint.—G. R. Pisek.
New York State Jour. Med., January, 1914.

Infant Feeding, Modern Methods of.—G. Fulton.
Kentucky Med. Jour., Feb. 1, 1914.

Malted Gruel for Sick Infants, Advantages of.—I. Rosenthal and H. P. T. Oerum.
Arch. f. Kinderh., 1914, lxii, Nos. 3-4.

Milk, Sanitary Significance of Body Cells in.—R. S. Breed.
Jour. Infect. Dis., January, 1914.

Milk, What is Genuine.—W. C. Dundas.
Edinburgh Med. Jour., January, 1914.

Nourishment during Pregnancy, and Weight of the Child.—J. Bondi.
Wien. klin. Wchnschr., Jan. 1, 1914.

Nutrition of Infants, Physiologic Principles Concerned in.—E. Pritchard.
Practitioner, London, January, 1914.

Scorbutus, Analysis of the Viscera in.—H. Bahrdt and F. Edelstein.
Ztschr. f. Kinderh., Jan. 31, 1914.

Scorbutus, Infantile.—W. Wernstedt.
Hygiea, January, 1914.

Whole Milk Dilutions in Feeding Normal Infants.—J. A. Gannon.
Washington Med. Ann., January, 1914.

DISEASES OF THE NEW-BORN

Hemorrhage, Gastro-Intestinal, in Two New-Born Infants Nursing Same Woman.—A. Bonnet-La-Dorderie.
Rev. méd. de la Suisse romande, Dec. 20, 1913.

ACUTE AND INFECTIOUS DISEASES

Air as a Vehicle of Infection.—Charles V. Chapin.
Jour. Am. Med. Assn., Feb. 7, 1914.

Cell Inclusions in Scarlet Fever and Measles.—E. H. Ross.
Jour. State Med., London, February, 1914.

Cerebrospinal Meningitis, Epidemic, Neurologic Aspects.—R. M. Van Wart.
New Orleans Med. and Surg. Jour., February, 1914.

Chorea, Chronic Progressive, Lesions of.—P. Marie and J. Lhermitte.
Ann. d. méd., January, 1914.

Diphtheria.—W. H. McLain.
West Virginia Med. Jour., January, 1913.

Diphtheria, Changes in the Hypophysis in Experimental.—S. Abramow.
Virchow's Arch. f. path. anat., 1914, ccxiv, No. 3.

Diphtheria, Effect of Lactic Acid Bacilli on.—S. T. Nicholson, Jr.
Jour. Am. Med. Assn., Feb. 14, 1914.

Diphtheria, Mechanism of Circulatory Failure in.—W. G. MacCallum.
Am. Jour. Med. Sc., January, 1913.

Glandular Fever or Follicular Diphtheria?—P. Galli.
Gaz. d. osp., Nos. 151-155.

Infantile Paralysis, Principles of the Treatment of.—Robert W. Lovett.
Jour. Am. Med. Assn., Jan. 24, 1914.

Infectious Diseases, Isolation and Quarantine Periods in.—C. B. Ker.
Edinburgh Med. Jour., January, 1914.

Influenza, Abdominal, in Boy of 13 with Temperature of 105.8 F.—J. Vergely.
Jour. d. méd. d. Bordeaux, Jan. 25, 1914.

Measles, Early Diagnosis of.—M. Grumann.
München. med. Wchnschr., Jan. 20, 1914.

Measles, Early Diagnosis of, Value of Blood Picture in.—W. P. Lucas.
Am. Jour. Dis. Child., February, 1914.

Meningitis and Public Health.—A. D. Patillo.
Texas State Jour. Med., January, 1914.

Meningitis Carriers.—J. H. Black.
Texas State Jour. Med., January, 1914.

Pellagra in Early Childhood.—W. Weston.
Am. Jour. Dis. Child., February, 1914.

Pertussis, Case of Atrophy of Optic Nerve after, Seventh on Record.—E. Rodelius.
Arch. f. Kinderh., 1914, lxii, Nos. 3-4.

Poliomyelitis, Diagnosis of Epidemic, and Treatment in Acute Stage.—Monrad.
Ugeskr. f. Laeger, Jan. 8, 1914.

Poliomyelitis, Experimental, Intraspinous Infection in.—P. F. Clark and H. L. Amoss.
Jour. Exper. Med., February, 1914.

Poliomyelitis, Epidemic, Etiology of.—H. L. Amoss.
Jour. Exper. Med., February, 1914.

Poliomyelitis, Epidemic, Etiology of.—F. Lust and F. Rosenberg.
München. med. Wchnschr., Jan. 20, 1914.

Poliomyelitis, Epidemiology of.—P. A. Lewis and A. G. Margot.
Jour. Exper. Med., February, 1914.

Poliomyelitis, Retention of Urine during Acute Stage of Epidemic.—G. Schreiber and D'Allaines.
Ann. de méd. et chir. inf., Jan. 1, 1914.

Scarlet Fever.—N. Witt.
Hygiea, December, 1914.

Scarlet Fever, Case of, in Child Two Weeks Old.—M. P. Rucker.
Old Dominion Jour. of Med. and Surg., January, 1914.

Scarlet Fever Convalescent Serum, Conservation of.—B. Koch.
München. med. Wchnschr., Dec. 30, 1913.

Scarlet Fever, Serotherapy in.—A. Cederberg.
Berl. klin. Wchnschr., Jan. 12, 1914.

Scarlet Fever, Serotherapy in.—Moog.
Therap. Monatsch., January, 1914.

Scarlet Fever, Administrative Measures for Control of.—J. J. Benton.
California State Jour. Med., January, 1914.

Septic Sore Throat Epidemic in Cortland and Homer, N. Y.—C. E. North, B. White and O. T. Avery.
Jour. Infect. Dis., January, 1914.

Small-Pox of Fetus in Fourth Month.—C. Hall.
Australasian Med. Gaz., Jan. 3, 1914.

Small-Pox, Some Aspects of, in Infants and Children.—W. Hanna.
Brit. Jour. Child. Dis., January, 1913.

Stomach Aches Associated with General Infections in Childhood.—H. T. Gray.
Practitioner, London, January, 1914.

Varicella, Nervous Complications of.—R. Miller and J. A. Davidson.
Brit. Jour. Child. Dis., January, 1913.

Why Schoolchildren Should Be Vaccinated.—O. L. Williamson.
Arkansas Med. Soc. Jour., January, 1914.

TUBERCULOSIS AND SYPHILIS

Anaphylaxis and Antianaphylaxis in Young Tuberculous Children and Relation to Tuberculin Treatment.—G. Di Cristina and G. Caronia.
Arch. f. Kinderh., 1914, lxii, Nos. 3-4.

Infection of Children with Bovine Tubercle Bacillus.—A. P. Mitchell.
Brit. Med. Jour., Jan. 17, 1913.

Meningitis, Cerebrospinal Fluid in Tuberculous.—Mandelbaum.
Deutsch. Arch. f. klin. med., Jan. 17, 1914.

Tuberculosis among the Schoolchildren at Odessa, Russia.—J. M. Arluck.
Arch. f. Kinderh., 1914, lxii, Nos. 3-4.

Tuberculosis, Diagnosis of Earliest Stage of, in Children.—H. G. Gade.
Norsk. Mag. f. Laegevidensk., January, 1914.

GASTRO-INTESTINAL SYSTEM

Alimentary Disease in Infant and Child, Bacteriologic Diagnosis and Treatment of.—R. Vincent.
Am. Jour. Dis. Child., February, 1914.

Diarrhea, Starch, in Infants.—Hutinel.
Presse méd., Dec. 27, 1913.

Expulsion of Entire Lining of Esophagus after Drinking Lye; Two Cases; Recovery in One.—E. Liebmann.
Med. Klin., Jan. 11, 1914.

Gastro-Enteritis, Bacillus Lactis Bulgaricus in Infantile.—L. H. Schwartz.
Med. Rec., New York, Jan. 24, 1914.

Gastro-Enteritus in Infants, Restriction of Fluids in Treatment of.—J. Anderodias.
Jour. d. méd. d. Bordeaux, Jan. 25, 1914.

Pyloric Stenosis, Congenital; Study of Twenty-two Cases with Operation by the Author.—H. M. Richter.
Jour. Am. Med. Assn., Jan. 31, 1914.

RESPIRATORY SYSTEM

Electro-Magnets, Use of, in Extraction of Metallic Bodies from Trachea and Bronchi, with Report of Cases.—S. Iglauer.
Lancet Clinic, Jan. 24, 1914.

Pneumonia, Significance of Plasma Cells in, in Children after Acute Infectious Diseases.—J. Watjen.
Virchow's Arch. f. path. Anat., Berlin, 1914, ccxiv, No. 3.

CIRCULATORY SYSTEM

Eosinophilia in Infants; a Symptom of the Exudative Diathesis.—H. Putzig.
Ztschr. f. Kinderh., Jan. 31, 1913.

NERVOUS SYSTEM

Anorexia Nervosa.—N. G. Orchard.
New York State Jour. Med., January, 1914.

Brain Tumors in Children.—L. d'Astros.
Ann. de med. et chir. inf., Paris, Dec. 24, 1913.

Epilepsy Based on One Thousand Admissions to Kansas State Hospital for Epileptics, Study of.—M. L. Perry.
Kans. Med. Soc. Jour., January, 1914.

Epilepsy, Metabolism in.—S. Kozlowski.
Ztschr. f. klin. Med., 1914, lxxix, Nos. 3-4.

Epilepsy, Therapeutic Value of Crotalin in Treatment of.—T. J. Mays.
Med. Rec., New York, Jan. 17, 1913.

Myatonia Congenita (Oppenheim), Case of.—J. W. Courtney and H. B. Eaton.
Boston Med. and Surg. Jour., Jan. 22, 1914.

Mental Defect in Children, Prevention of.—T. Thompson.
Jour. State Med., London, February, 1914.

Mental Deficiency, Diagnosis of, in Infancy and Childhood.—H. H. Donnally.
Am. Jour. Obst., January, 1914.

Spasmophilia with Special Reference to Familial Reaction and Repeated Absences; Report of Sixteen Cases.—J. P. Sedgwick.
Am. Jour. Dis. Child., February, 1914.

GENITO-URINARY SYSTEM

Blood-Pressure in Children Showing Orthostatic Albuminuria.—H. M. Bass and H. Wessler.
Arch. Int. Med., January, 1914.

Kidneys, Granular Atrophy of, in Children.—C. Jacobs.
Berl. klin. Wchnschr., Dec. 29, 1913.

OSSEOUS SYSTEM

Osteochondritis Deformans Juvenilis.—D. P. D. Wilkie.
Edinburgh Med. Jour., January, 1914.

SKIN AND APPENDAGES

Psoriasis in Infancy.—H. Davis.
Brit. Jour. Child. Dis., January, 1913.

EYE, EAR, NOSE AND THROAT

Pharyngeal Lymphoid Ring, Relation of, to General Health.—H. C. Markham.
Kans. Med. Soc. Jour., Jan., 1914.

Tonsils, Actinomyces-Like Granules in.—D. J. Davis.
Jour. Infect. Dis., January, 1914.

Tonsils, Hypertrophied.—E. Wilson.
Kentucky Med. Jour., January, 1914.

Tonsil Operations on Public School Children in New York City, Results of.—G. H. Cocks.
New York Med. Jour., Jan. 17, 1913.

Tonsillectomy, Method of, by Means of Alveolar Eminence of Mandible.—C. A. Gundelach.
Iowa State Med. Soc. Jour., January, 1914.

Tonsils, Removal of Pair of Extraordinary Size.—B. C. Gile.
Pennnsylvania Med. Jour., January, 1914.

Trachoma.—C. L. Zugg.
Kans. Med. Soc. Jour., January, 1914.

SURGERY AND ORTHOPEDICS

Fractures in Children.—L. Schooler.
Iowa State Med. Soc. Jour., January, 1914.

Hip, Congenital Dislocation of; Rational Method of Treatment.—H. M. Sherman.
Surg., Gynec. and Obst., January, 1914.

Hip Joint, Congenital Dislocation of.—K. Ludloff.
Berlin. Klin. Wchnschr., Jan. 19, 1914.

Orthopedic Surgery, Recent Advances in.—E. W. Ryerson.
Texas State Jour. Med., January, 1914.

Spine, Abbott's Treatment for Curvature of the.—L. Ombrédanne.
Presse méd., Jan. 3, 1914.

Spinal Curvature.—R. E. Roth.
Australasian Med. Gaz., Dec. 27, 1913.

Spinal Lateral Curvature, Correction of the Fixed Structural Type of.—Roland O. Meisenbach.
Jour. Am. Med. Assn., Feb. 14, 1914.

Weak Feet, Importance of Treatment of, in Childhood.—B. H. Whitbeck.
New York State Jour. Med., January, 1914.

THERAPEUTICS

Mouth-Drip. A Method of Administering Fluid to Infants in Gastro-Enteritis.—Alfred F. Hess.
Jour. Am. Med. Assn., Chicago, Feb. 7, 1914.

MISCELLANEOUS

Boy of 13 Never Learned to Swallow Solid Food.—T. Goett.
Ztschr. f. Kinderh., Berlin, Jan. 31, 1913.

Call of the Child.—Frances Bradley.
Jour. Am. Med. Assn., Chicago, Jan. 24, 1914.

Dentition, Helminthiasis and Growth as Scape-Goats.—Gaujoux.
Ann. de méd. et chir. inf., Jan. 1, 1914.

Disorders in Children by Imitation and Induction.—A. Strauch.
Med. Rec., New York, Jan. 17, 1913.

Infant Mortality, Rôle Played by Obstetrician in Prevention of Infant Mortality.—A. F. Furrer.
Cleveland Med. Jour., January, 1914.

Influence of Water-Borne Disease on Mortality of Children.—A. J. McLaughlin.
Amer. Jour. Trop. Dis. and Prevent. Med., January, 1914.

Mother and Child Are Independent Biologic Entities.—R. v. Fellenberg and A. Döll.
Ztschr. f. Geburtsh. u. Gynäk., Jan. 10, 1914.

Pediatric Literature of 1913, Review of.—O. N. Torian.
Indiana Med. Assn. Jour., January, 1914.

Proctoclysis for Children.—E. Lesne.
Ann. de méd. et Chir. inf., Jan. 1, 1914.

Speech, Demonstration of Defects of.—G. Hudson-Makuen.
Pennsylvania Med. Jour., January, 1914.

Summer Infant Mortality.—L. Nicolaysen.
Norsk Mag. f. Laegevidensk., January, 1914.